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"Scire est nescire, nisi id me
Scire alius sciret."

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THE LONG FOX MEMORIAL LECTURE:

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THE VICE-CHANCELLOR (Dr. T. LOVEDAY, M.A., LL.D.) *in the Chair.*

BY

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ON

THE PARTNERSHIP BETWEEN ANÆSTHESIA AND SURGERY.

As a product of the Bristol Medical School, naturally I am fully sensitive of the privilege which the Committee have bestowed upon me, and I wish to offer to them my sincere thanks for inviting me to deliver this address. Nevertheless, I am painfully alive to my responsibility in undertaking so active a part in this the twentieth annual meeting held to honour

the name of the late Dr. Long Fox. The name is one that Bristol is justly proud of, and stands for a rare combination of scientific thought and untiring energy devoted to clinical services and benevolent actions. Owing to the fact that he made a special study of nervous diseases in general, and of those connected with the sympathetic system in particular, it seems probable that, had he been living to-day, Dr. Fox would have been interested in the subject of anæsthesia, seeing how intimately connected some of its phenomena are with the functions of the involuntary nerves.

When a medical student I once had the privilege of being present during a consultation on a patient with symptoms of raised intracranial pressure which Dr. Long Fox diagnosed as due to a cerebral tumour. He expressed the view that trephining might relieve the distress, but that the patient would probably be unable to stand the necessary anæsthetic, which would undoubtedly have been chloroform. Little did I think, on that occasion, that at a future date, forty years thence to be exact, it would fall to my lot to discuss with you, under the ægis of Dr. Fox's memory, the problem of making bad risk operations safer, and the importance, to both surgery and anæsthesia, of the clearest understanding between the specialists concerned.

We live in an age of rapid progress, and may easily forget how far we have travelled or what we have left behind us, but a comparison of the conditions under which operations were performed in pre-anæsthetic days with those obtaining to-day will help us to appreciate the enormous strides which surgery has been able to make since her association with her faithful handmaiden, anæsthesia.

I cannot think that any useful purpose would be served by an attempt on our part to visualize in detail all the horrors inseparable from surgery in those days, or the terrible scenes witnessed in operating theatres so graphically described by many authors,[1] but we can well imagine that before the introduction of anæsthetics operative surgery must, indeed, have been a brutal and nerve-racking occupation; a most unlikely parent, one would have thought, for an offspring so sympathetic and kindly as the operative surgery of our acquaintance. It is interesting to note from what depths of ill-favour surgery has been raised to its present high position in the world of science. As evidence, we read that Abernethy regarded operations as having no place in surgery as a science; also that in 1753 Hunter was strongly advised to study medicine rather than surgery, as being a higher branch of the healing art, and he himself is alleged to have regarded the surgeon as a form of armed savage.

The feelings of the time found expression, in 1774, in the hostile attitude of the students of Freiburg towards von Wüthevehr, whom they threatened to mob for daring to advocate the union of surgery with medicine. About this time schools of surgery were established for the benefit of the army, but in Germany it was still considered the duty of surgeons to shave both officers and men, although in England, in 1745, an Act of Parliament was passed to abolish the degrading association of barbers and surgeons.

It seems almost incredible that under such conditions men could be found to inflict, or patients to bear, the agony which an operation must have involved, and we learn that even such a stalwart as Cheselden could seldom sleep on the night preceding

an operation. Some idea of the suffering to which a patient had to submit may be gathered from a letter which Simpson received from a colleague describing his own experience when under the knife. He says: "God and man deserted me, and a black whirlwind of emotion and the horror of great darkness overwhelmed me."

In spite of the existing difficulties quite a lot of surgery was performed, nor was it confined to operations of an emergency type. In the seventeenth century, for instance, Abraham Cyprianus, of Amsterdam, besides doing lithotomy no less than 1,400 times, performed abdominal sections and radical cures for hernia; and Larrey, who is said to have conducted 200 amputations in one day after the battle of Moskowa, claimed, among his successes, a case of removal of loose cartilage from a knee-joint, the wound healing by first intention.

I have not quoted the above facts for their historical interest, but in order to illustrate what was the potential eagerness pent up in surgery ready to expand directly the restraining influence of pain and sepsis could be removed.

With the whole world so urgently in need of some method that could assuage pain and thus render surgery available for everybody, it seems surprising that more notice was not taken of the famous letter written in Bristol by Humphry Davy in 1800, in which he said: "As nitrous oxide appears capable of destroying physical pain, it may probably be used with advantage in surgical operations in which no great effusion of blood takes place."[2] That this valuable suggestion should have been allowed to pass unnoticed was due perhaps to the fact that the invention does not seem to have been demonstrated

at any surgical centre, and illustrates the truth of our Medico-Chirurgical motto: "Scire est nescire, nisi id me scire alius sciret," or in other words: "One might as well not know a thing as know it and keep it to oneself." On the other hand, however much we may regret the failure to publish more widely this discovery of the properties of nitrous oxide, we must remember that it was not until 1870 that the gas was obtainable in a compressed state in portable cast-iron cylinders, so that its adoption in surgery would have necessitated the centralization of operative work at institutions where the gas could be made on the premises.

The efforts of Colton, begun in 1863, to popularize the gas in dentistry, failed to meet with much success until the year 1867, when he actually demonstrated the method to an English dentist practising in Paris, and from that moment its popularity quickly spread.

In the meantime the use of ether anæsthesia has gone through the same curious phase of remaining for some years known but not appreciated. Its real introduction to the world dates from the occasion in 1846 when Morton administered ether to a patient, on whom Dr. Warren operated, at the Massachusetts General Hospital, but in a letter to Dr. Warren concerning experiments which he had made with ether at the Massachusetts Hospital ten years earlier, in 1836, Morrill Wyman writes: "We each tested the ether by breathing it from a bottle till it produced unconsciousness, the others watching the different effects upon each. We also experimented upon rats in a glass globe until they were entirely motionless, and often wondered that the treatment did them no harm. But we never thought of trying the sensibility under

ether even by pricking with a pin. It was a great oversight."

The same thing occurred in reference to chloroform. Early in 1847 Flourens described its power to produce anæsthesia in animals, but its effect was not tried upon the human subject until some months later, when Simpson experimented upon himself during his search for a more pleasant drug than ether; he soon made known his discovery, and within a short time, as the use of the drug spread in many quarters, disasters began to occur under its influence. The mysterious and dangerous phenomena associated with chloroform narcosis have attracted the attention of workers in a variety of research fields, with the ultimate result that the subject of anæsthesia is becoming daily more logical and better understood by both surgeons and administrators.

These three drugs—chloroform, ether and nitrous oxide—together with certain hypnotics and analgesics, form the armamentaria with which the anæsthetist endeavours to co-operate with the surgeon in a combined effort to prevent surgical shock and to avoid post-operative complications as far as possible; in fact, to leave the patient, after operation, in a condition as nearly normal as circumstances will permit. But the surgeon and anæsthetist, in their search for this goal, will have to follow paths set so close together that it will be necessary for them, in places, to proceed, metaphorically, hand in hand, with the possibility of occasionally treading on each other's toes.

Ordinary cases can be adequately dealt with on routine lines; but the bad risk operation is generally preceded by a consultation between all concerned, and it is no unusual thing to find in such cases that the patient stands the ordeal much better than was

expected, leading one to speculate whether the good result which so frequently follows such consultations should be attributed to a modified anæsthesia or to a moderated surgical trauma.

We may be excused if we seem over-ready to blame the surgeon for the evil effects of trauma, when with justice we should rather turn to the failure of our anæsthetic to subdue reflexes and counter the shock caused by manipulation; but we may have been deterred, on the one hand, from using an adequately deep anæsthesia owing to our fear of the depressing result of an over-dose, or we may have omitted to use or to suggest the use of an analgesic. This represents a typical example of the overlapping of responsibilities. As a specific illustration of such overlapping I might quote from my notes of a series of thirty-six abdominal operations performed by the same surgeon, in which a combination of light anæsthesia with gentle manipulation resulted in a remarkable freedom from after-effects, except, however, as regards three of the patients, who vomited and showed symptoms of shock, and in these three the abdominal cavity had been "packed off" with towels of a thick, harsh fabric, quite different from the material used for this purpose in the remainder of the series; I feel that this was an instance of faulty partnership, and that either I ought to have deepened the plane of anæsthesia or the surgeon should have employed an analgesic or softer cloths so as to mitigate trauma.

As a matter of fact, the problem of avoiding shock has yielded in a most satisfactory manner to modern devices, but there has not been the same degree of improvement as regards chest complications; and to introduce the topic before a gathering of this nature,

with surgeons present, and where discussion is not expected, may be likened to waving a red rag in front of a tethered bull. In reference to anæsthetic risks Sir Benjamin Richardson held the conviction that there was a section of the community, about 1 in every 3,000, which consisted of people ever ready and willing to die, whom he designated as the "Morituri." Although we may hold a more optimistic view nowadays of the attitude of patients to surgery, nevertheless, an analysis of anæsthetic statistics[3] may well leave us inclined to extend this classification so as to include another section consisting of people always ready to develop pneumonia or bronchitis, to be defined as the "Expectoraturi." It will be agreed that this type of individual, when requiring high abdominal surgery, presents us with some of our most difficult problems, demanding the very closest co-operation between surgeon and anæsthetist, in order that the two may realize that they are complementary to one another; in other words, that the more the former assists by limiting the amount of trauma and of heavy stimuli, the less likely will the latter be to cause damage by administering too much anæsthetic.

The other complication most commonly met with after inhalation anæsthesia is sickness, and is generally attributable to the anæsthetic, although in certain cases it may be the result of surgical conditions, such as peritonitis or distended and paralysed intestine. There are, however, a few people who seem to be almost incorrigible in their tendency to vomit, and who might almost claim yet another place in Richardson's classification; but, with modern methods of anæsthesia the incidence of this sequela has dwindled to comparatively small proportions, a result due

partly to less anæsthetic poisoning and partly to improved pre-operative preparation, especially as regards the giving of more nourishment, and the avoidance of drastic purging.

The interest taken in the properties and action of the inhalation anæsthetics by workers in various fields of research has resulted in the accumulation of a mass of valuable data upon which to base the choice of method to be used in any particular case, and the selection will depend upon the following factors: safety during administration, capacity as a relaxant, degree of toxicity, reversibility, pleasantness, and ability to prevent shock. Chloroform provides the best form of anæsthesia of any inhalant, keeping the patient perfectly relaxed and immobile, and its abandonment as a routine anæsthetic must have entailed a heavy sacrifice of comfort on the part of operators, but such a course was inevitable in the interest of the patient.

The first reported death under chloroform, which occurred in January, 1848, in Hanna Greener of Newcastle, was soon followed by many others, with the result that an interest in the subject grew up among the profession and spread to other scientists, including physiologists, pharmacologists and chemists, amongst whom Professor Buckmaster held a prominent position.[4] This interest has, fortunately, never been allowed to flag, but has been extended to every problem connected with anæsthesia up to the present time. Between the years 1864 and 1891 no less than four special commissions were convened to discover the causes and means of avoiding danger with chloroform. The outstanding feature of this drug is undoubtedly its tendency to produce a depressant effect upon the circulatory and respiratory systems,

and its poisonous action on the heart muscle either when given in concentrated strength for a short period or in a weak form over a prolonged period. This characteristic makes its safety in administration dependent upon the closest attention to detail, and immediate cessation of operation and institution of remedial measures at the first sign of danger. This can only be possible where there is agreement between surgeon and anæsthetist, delay of a few seconds easily leading to death.

The beautiful relaxation so characteristic of this drug kept it in favour for abdominal surgery in spite of the unenviable reputation it was winning for itself in general, but its popularity received a lasting set-back when in 1903[5] Guthrie proved beyond doubt the toxic effects which could be caused by it. The fact soon became recognized that this danger was inseparable from the use of chloroform, and accounted for the persistent nature of the vomiting which was wont to follow a prolonged anæsthesia under its influence. Surgeons were quick to respond by insisting that the drug should not be employed in cases where the integrity of the liver was under suspicion, some even going so far as to exclude it from their clinics altogether. As long ago as 1915 Mr. Moynihan, as he then was, told me that he refused to allow chloroform to be used in any form in his private nursing home.

In 1897, when Mr. Tyrrell introduced his two bottle Junker apparatus for chloroform and ether, he claimed as one of its advantages a reduction in the frequency of vomiting. In the years 1910 to 1912 Mr. Stock and I, in the Infirmary, found that vomiting and acetonuria varied in proportion with the amount of chloroform used. The strength of vapour was controlled by using Mr. Stock's apparatus, when ether

vapour was to be added, otherwise by means of the Vernon Harcourt machine, or by a drop method entailing the counting of each drop. The light plane of anæsthesia resulting from this reduction to a minimum of the amount of anæsthetic vapour administered tended to allow the breathing to become too shallow. This symptom was relieved, in a few instances, by the administration of oxygen with 5 per cent. CO_2, as had recently been shown by Henderson[6] to be feasible. The result of these investigations went to show that vomiting and acetonuria varied in direct proportion. In a small series of cases in which I had the assistance of Professor Walker Hall in testing for acetone in the blood and urine of the patients in whom the anæsthetic plane had been kept light the incidence was 2 per cent. compared with 10 per cent. in cases where the corneal reflex had been kept in abeyance throughout.

In 1876[7] Claude Bernard stated that all things living were capable of being anæsthetised, and bearing out this suggestion, a good deal of experimental work has been conducted on lower organisms in order to test the relative toxicity of the different anæsthetic drugs, especially of chloroform, ether and nitrous oxide. In 1922 Mr. Hunter and I,[8] working in the botanical and biological departments of this University,[7] were able to prove, to our complete satisfaction, that in every organism we dealt with the toxicity of chloroform was infinitely greater than that of ether, the stimulating phase was shorter and less pronounced, and the paralysing effects were more marked and more slowly recovered from; these results seem to correspond with the phenomena met with clinically.

The paralysing effect produced by chloroform upon

the muscles of the abdominal wall naturally meant a degree of relaxation which rendered manipulation easy and free from trauma, and led to the belief that this drug had a special property for preventing shock; but as this involved an undesirable degree of toxicity, it became obvious that the disadvantages outclassed the advantages, with the result that the drug gradually came to be replaced wholly or in part by other agents.

Towards the end of last century Sir Benjamin Richardson, speaking with an intimate knowledge of the action of anæsthetic agents, expressed the opinion that general anæsthesia must eventually give way to local. The tendency to-day is in that direction.[9] The replacement of chloroform by the less toxic ether, and later the attempt to substitute for ether the less poisonous nitrous oxide, may be regarded as parts of the scheme. In this Bristol centre the subject was brought before the profession by Dr. John Freeman at meetings of the Medico-Chirurgical Society in 1895 and 1896. In this neighbourhood, as elsewhere, it was not long before surgeons yielded to conviction.

It may not be unreasonable to claim for ether that it has proved itself a really important factor in the recent advances of surgery. By reason of its safety and flexibility it can, within certain limits of time, be administered in strengths sufficient to subdue or abolish deep reflexes which, if allowed to remain active, would lead to the development of shock and by this means extensive operations may be rendered comparatively harmless. It can be used in practically every operation and in almost every type of patient.

When employed in a closed inhaler ether enables the administrator to exercise with ease complete and instant control over the depth of anæsthesia, but after a time it is apt to increase the movements of

respiration and the secretion of mucus to such an extent that abdominal relaxation is no longer possible to obtain. This characteristic led to the adoption of either the open or the perhalation method of administration.

The effects produced by these two methods differ in important essentials, which must be carefully considered before deciding which to employ. The perhalation system, by yielding a deeper and more typically etheric form of anæsthesia, is the easier from the point of view of the administrator, but has the drawback of delivering a vapour chilled below the temperature of the surrounding air, whereas with the frankly open method the reduction of temperature is negligible. In the opinion of many experienced anæsthetists the incidence of lung complications following open ether is at least as great as that associated with closed methods, but these statistics are vitiated by the fact that the term "open ether" is generally used in reference to the perhalation system.

The strictly open method gives a narcosis too light for abdominal surgery in muscular subjects, and may need to be subsidized by the injection of an analgesic, or by the use of pre-operative medication in order to secure relaxation; but it has the advantage of leaving the patient only slightly saturated with the drug, and therefore not prone to prolonged sequelæ. My reason for dilating in some detail upon this part of my subject is, not because I intend to bring forward the usual statistics, proving the superiority of one form of administration over all others, but because my experience in some thousands of cases has taught me, above all things, the inestimable value of the surgeon's co-operation. Where gentle manipulation is not

possible, or where a hypnotic or local analgesic is not available, the method may prove unsuitable for abdominal work.

Speaking generally, ether may be said to afford good protection against surgical shock; but this term is a comprehensive one, means any degree of lowered vitality following operation, and should be understood to include all states and degrees of lowered blood-pressure, reduced temperature or impaired resistance to infection. The cause of shock may lie either in surgery or in anæsthesia, and the remedy be a question for consideration of the partnership.

From the anæsthetist's point of view, the course to be taken lies between his Scylla and Charybdis. Too light a narcosis leads to shock by rendering gentleness of manipulation impossible, and too deep an anæsthesia may produce shock through the poisonous effect of the drug itself. In a series of 100 cases at the Bristol Royal Infirmary in 1906, during an investigation into the respective values of the deep and light types of anæsthesia, it was found that the average loss of temperature after operation was invariably greater after deep than after light narcosis, the degree of anæsthesia being determined by the absence or presence of the corneal reflex. On a subsequent occasion, in 1922, Dr. Todd kindly investigated the opsonic index at intervals during ether administration; in each case, after an initial small rise, there was a drop of from 20 to 30 per cent. after ether had been administered for half an hour. This effect upon the phagocytic activity of leucocytes was described by Hamburger[10] in connection with chloroform, and by Graham[11] in connection with ether.

The above clinical experiences have been quoted

here because they agree with the findings of certain experimental workers.

Dr. Frank Mann,[1 2] of the Mayo Clinic, investigating the effect of various ether vapour tensions upon vascular reflexes, found that the best tensions for operative work, in the dog, lay between 38 and 45, and that the corneal reflex was very active with tensions between 36 and 40, but never persisted with tensions over 45. He found that the blood-pressure was always markedly depressed when the ether tension reached 48 or 50. He adds that with the lower tensions, 36 to 45, any depression is quickly recovered from, even after the animal has been kept under ether for some hours; but that with the higher tensions, 48 to 55, the blood-pressure is slow to recover after the ether is withdrawn and the animal allowed to "come round."

The tendency of ether inhalation to be followed by chest complications makes it advisable that in certain types of operations its use should be limited as far as convenient; it is in this connection that nitrous oxide has proved of such great service. It seems almost incredible to-day that at one time this gas, N_2O, was thought to be a poison of the most virulent nature. In *The Considerations on the Production of Fictitious Airs*, published by Dr. Beddoes, 1794–1796, a paper on nitrous oxide appeared, in which the author, S. Latham Mitchill, stated that this gas was so poisonous that a single inhalation of it would destroy life at once, and that it was actually the virus of the fevers and plague. In spite of this warning Davy had the courage to test the truth of the statement by inhaling one deep inspiration of the product, and being unaffected was thus assured that the allegation was false.

The reputation for safety which gas anæsthesia enjoys in short cases does not apply with equal force in the case of prolonged administration, there being a tendency for progressive anoxæmia to develop in spite of the addition of oxygen.

The subject was scientifically investigated by Brown, Lucas and Henderson,[13] who drew the conclusion that "surgical anæsthesia (*i.e.* relaxation) cannot be produced by N_2O and O_2 under pressure up to two atmospheres, that surgical anæsthesia with N_2O depends on anoxæmia as well as cerebral depression, that anoxæmia is unavoidable, and that this must limit its use and increase its dangers." Even Davy, during the many months he spent investigating the properties of nitrous oxide, worked out the percentage of oxygen necessary to be mixed with nitrous oxide in order to sustain life, and found that there was a limit to the length of time any animal could be kept alive in this gas. These experiments were conducted with great care, the animals being passed through a medium of water or mercury into the jar containing the gases, so that the percentage of these should not be vitiated by the introduction of air carried in by the animals' fur. Davy assures us that the immersion caused the animals no, or only temporary, inconvenience !

In our own early experiences with nitrous oxide we were puzzled by this anoxæmia, it being easy to keep patients fully anæsthetized for a while, but obviously dangerous to attempt to maintain relaxation beyond ten minutes or so even with a liberal supply of air or oxygen.

In 1906 I used gas and air in a number of cases of midwifery, but found it impossible to produce, with safety, sufficient relaxation during the second stage.

Patients, however, were allowed to help themselves to the gas during pains, and trouble never arose from this arrangement.

It is important that the partnership should agree on this point, otherwise the surgeon may expect deep anæsthesia to be kept up without the assistance of premedication, or local analgesics, or ether. Even with ethylene and oxygen in equal proportions some ether is required for complete relaxation.

Before embarking on a gas anæsthesia the administrator should understand how much he is to look to ether or to a preliminary hypnotic and how much to a nerve blocking for the necessary assistance.

Crile, in expounding his famous anoci-association method, suggested that the surgeon who finds relaxation unsatisfactory should look to his local analgesia rather than to the anæsthetist for rectification of the fault. I would suggest that the surgeon should not hesitate to tell the anæsthetist what is needed, remembering that nothing hurts the feelings of the anæsthetist more than a profane silence on the part of the surgeon; it signifies a state of tension in the partnership.

As regards the frequent occurrence of lung complications after high abdominal operations, although we must attribute a large portion of the blame to the anæsthetic, we cannot afford to overlook the element of paralysis affecting the nerves which govern the movements of the lower intercostals and diaphragm, and at the same time innervate the peritoneum covering the gall-bladder, the diaphragm and the pyloric end of the stomach, and one is led to wonder whether there will ever be discovered a surface

anæsthetic with so low a toxicity that its application to a large surface such as that of the peritoneum will be safe. Percaine may bring us a step farther in this direction. Such a drug would revolutionize anæsthesia in many respects.

In connection with local anæsthetic drugs the work done in this University some years ago by Professor Francis and the experiments which Mr. Eric Watson-Williams[14] conducted here in 1925 represent the sort of investigations from which we may hope, some day, to derive such a boon to surgery.

The subject of basal anæsthetics is much to the fore at present, and the possibilities of developing the chemistry of the barbiturates along rational lines has been explained in this Department as recently as March, 1931, by Dr. Nierenstein, with demonstrations by Professor Brocklehurst,[15] with an account of their pharmacological aspects by Mr. A. L. Taylor. The wholesome result of such scientific help to the profession is that it enables us to realize how desirable it is that we should try to include in our partnership workers in academical departments.

In England to-day we occupy a particularly fortunate position as regards anæsthesia, with ether and gas holding the central position and with hypnotics and local analgesia, including spinal, well established, so to speak, on either side. It is true that manufacturers report that there still exists a large demand for chloroform, but as far as the large teaching clinics are concerned it is used chiefly as an auxiliary, and chloroform anæsthesia proper has become very rare.

Local methods are coming into their own, but in a country like this, where there are so many expert inhalation anæsthetists, such methods can be kept

for occasions where they offer some real advantage to the patient, and where less tedious methods are contra-indicated for some reason.

The decision as to which type of anæsthesia to employ is at times extremely difficult, especially as in desperate cases the problem of risk is apt to be viewed from opposite angles by the surgeon and the anæsthetist. The question is often asked: "Can the patient stand an anæsthetic?" But seeing that there is practically no condition in which some form of anæsthetic cannot be taken, it might be more logical to inquire whether the patient could survive an anæsthetic plus the proposed operation. In other words, we have to remember that anæsthesia does not offer a complete barrier to the shock of surgery.

The same difficulty arises when spinal analgesia is advocated in such cases. There is a tendency to forget that a patient may be too ill for spinal and yet able to take a general anæsthetic with comparative safety.

May I tender my thanks to you, Mr. Vice-Chancellor, for so kindly presiding over this meeting, and to my audience for so patiently listening to me and for so politely remaining in the stage of consciousness; and explain that in order to attune my remarks, as far as possible, to the spirit which I believe should be displayed in these lectures, I have omitted the optimistic accounts of new drugs and infallible methods coming to us almost daily from outside, and have attempted to produce a few impressions based upon work done in home circles.

I feel that Dr. Long Fox would have preferred us to relate what we ourselves have done, rather than what we may have heard or read from outside sources.

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SILICOSIS.

I.—ADMINISTRATIVE AND CLINICAL.

BY

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THE importance of recognizing chronic interstitial pneumonia or pulmonary fibrosis has increased of late because some forms are occupational in origin and may give rise to claims for compensation. Special medical schemes have been set up under the Workmen's Compensation Acts to deal with the compensation of workmen in certain industries.

Fibrosis of the lung may be classified as (*a*) pure fibrosis, *i.e.* no tuberculosis; (*b*) tuberculo-fibroid, *i.e.* tuberculosis becoming fibroid; (*c*) fibro-tuberculous, *i.e.* fibroid becoming tuberculous.

The distribution of the fibrosis may be local or diffuse.

1. *Local.*—This occurs in tuberculosis neoplasms, aneurysms, infarcts, compression of bronchi.

2. *Diffuse.*—This occurs in tuberculosis (but is apt to be unilateral), as a sequel of massive type of lobar pneumonia, after broncho-pneumonia (especially in measles, whooping-cough and influenza). In any type of recurrent bronchitis or broncho-pneumonia; in cases of asthma; and as result of inhalation of irritant

gases. It may be due to syphilis, to thickened pleura, or lastly to pneumoconiosis.

Pneumoconiosis (Greek κονίω = I cover with dust).—This name implies that the fibrosis is due to dust-inhalation. Many dusts can irritate the lungs; but dusts composed largely of organic material require—apart from infections they may convey—long periods of time (twenty to thirty years) before they produce any demonstrable changes in the lungs. Dust heavily laden with silica (SiO_2) may produce extensive changes within two or three years (particularly if combined with an alkali; clay and coal-dust seem to retard the action of silica). It may be said that the danger of any dust is in direct proportion to the percentage of its silica content. We may, therefore, devote our time to the consideration of silicosis.

INDUSTRIES IN WHICH SILICOSIS OCCURS.

1. Refractories industries, *i.e.* manufacture of ganister and silica bricks for metallurgical and other high temperature furnaces.

2. Sandstone industries, quarrying and dressing sandstone.

3. Granite industries, quarrying and dressing granite.

4. Slate industries, quarrying and dressing slate.

5. Metal-grinding, where steel instruments are ground on sandstone wheel, especially in "facing" a new grindstone before use.

6. Sand-blasting of metals.

7. Pottery manufacture. Ground flint is used to embed the articles of ware whilst they are being fired.

8. Silica crushing industries in which ground silica is used.

9. Tin mining, drilling silicious rock.

10. Coal mining, cutting through silicious stone to reach coal, and "dusting."

11. Asbestos industry; asbestos is magnesium silicate.

In these industries specialist medical boards have been appointed to make periodic examinations of the workmen both at entry and afterwards, also to examine for the issue of compensation certificates. The approach to these boards by individual claimants for compensation is via the regional medical officer. If he gives a certificate the claimant is examined by the board after the deposit of 30s.; if the regional medical officer refuses a certificate the claimant may still appeal to the board on depositing £8, of which £6 10s. is returned if the appeal succeeds.

SYMPTOMATOLOGY.—Silica dust may be invisible and is usually non-irritating, so that workers are unaware of the danger of inhalation.

Chronic form.—The first symptom is dyspnœa after many years' exposure in most cases, and it appears at first only on exertion. Apart from dyspnœa the symptoms are those of any respiratory disease, and depend on the character and degree of secondary infections and consequent changes. A French writer* has said: "The diagnosis of pulmonary silicosis is very difficult. It rests above all on the etiological data and on certain radiographic signs which are far from clear and require to be determined. In fact, in subjects presenting the signs of pulmonary sclerosis one thinks of silicosis when they have worked for some time in an industry where silica is present." In advanced but uncomplicated cases of general fibrosis of the lungs

* Policard, Magnin and Martin, *La Presse Medicale*, 1930, xxxviii. 875.

there may be a striking contrast between the extensive pulmonary lesions and the absence of marked constitutional symptoms.

Acute form.—A few cases after only two and a half to four years' exposure have manifested an acute form of silicosis. They first complained of dyspnœa. Then general constitutional disturbance developed with pyrexia, and an acute illness ended fatally in a few weeks. In these cases cyanosis was pronounced, and the whole picture resembled miliary tuberculosis.

DIAGNOSIS OF SILICOSIS FROM OTHER CHRONIC DISEASES WITH FIBROSIS.

The symptoms and course as stated by the patient and relatives are most important. Particular attention must be paid to histories of previous pulmonary ailments like asthma, bronchitis, influenza, pneumonia, tuberculosis and exposure to irritant gases. The early symptoms are :—

Dyspnœa on exertion.

Cough, usually short and paroxysmal, occurring at night and on rising in the morning. As a rule, a little viscid sputum like an oyster is eventually expectorated.

Cyanosis, quite slight as a rule, but a valuable sign if present.

Hæmoptysis, but this generally indicates a tuberculosis infection.

Pleuritic pains are common and appear early.

Clubbed fingers are rare, and suggest bronchiectasis, bronchiolectasis or tuberculosis.

PHYSICAL SIGNS.

Loss of weight is the rule, but it is not always rapid ; the general nutrition is below normal and the muscles are poor in bulk and tone. The chest expansion is

diminished, but the measuring tape is unreliable. The vital capacity should be measured by means of a spirometer. In shape the chest is flat in front and round-shouldered behind. The infra-clavicular fossæ are hollowed, and the intercostal spaces are indrawn; there is sometimes marked recession on inspiration. Above the clavicles the apices of the lungs may appear to be drawn down in inspiration, not flat and fixed as in tuberculosis, because there are not the apical pleural adhesions of tuberculosis. The accessory muscles of respiration are in action. The skin is pale, and subcutaneous venules appear on the chest.

Precordial pulsation is well marked, because retraction of the edges of the lung leaves the heart uncovered.

Tactile fremitus is, as a rule, increased.

Percussion.—As the changes are bilateral no contrasts in resonance are obtained. The note is higher pitched than normal, and the superficial area of cardiac dullness is increased by "uncovering."

The apices give a more resonant note than the lower areas, and the area of root impairment is enlarged, but differing from old hilar tuberculosis, in that there is impairment above and below the root dullness.

Auscultation.—Poor air entry is the rule. The respiratory murmur is weakened, especially on the right side and at the base. It may have a blowing character over the upper part of the chest which gradually spreads to the whole lungs.

There may be a few fine rales and clicks in the root areas, in the axillæ and bases, but the presence of rhoncus, sibilus and mucous rales is not characteristic of silicosis depending rather on associated bronchitis or bronchiectasis.

In the right mammary region about the fourth space the breathing is often puerile or almost bronchial. Dry pleural friction is frequently heard and felt in advanced cases, usually in the right axillary line to begin with. Vocal resonance is increased particularly towards the apices, where whispering pectoriloquy may also be well heard.

This description applies only to cases uncomplicated by tuberculosis or bronchiectasis. The presence of these complications will introduce the innumerable physical signs by which they betray themselves.

The most important aid in the diagnosis of the fibrosis of the lung is, however, radiography, with which subject Dr. Bush has kindly undertaken to deal.

SILICOSIS.

II.—RADIOLOGY AND PATHOLOGY.

BY

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I hope to be able to show you that a study of silicosis from two angles, the pathological and the radiological, is of more than academic interest, for in radiology we have a method, by means of serial X-ray examinations at different stages of the disease, of watching the pathological processes at work during life, and of correlating the findings with clinical symptoms and the degree of disability.

In a case suspected, on clinical grounds, of silicosis, the two deciding points are (1) a history of exposure to silica dust, and (2) X-ray findings. Either alone is not enough. If the disease is complicated by infection, such as tubercle (as it often is) the matter becomes more difficult; but in uncomplicated silicosis in the second or third stages, as I shall point out, the X-ray appearances are fairly characteristic.

Before we consider what actually occurs in the lung a brief survey of the *lymphatic system of the lungs and pleura* is at first necessary, since it is this system which is primarily affected in this disease. So far as we know anything about them, the following are the main points. There are no lymphatics in the alveoli themselves. Small nodules of lymphoid

tissue are found in intimate relation with the distal ends of the terminal bronchioles. There are two main groups of lymphatic vessels, deep and superficial. The deep lymphatics start at the terminations of the smallest bronchioles, and run in complex interlacing channels along with the bronchi and blood-vessels to the tracheo-bronchial and paratracheal glands at the roots of the lungs. The superficial lymphatics, which are said to have no anastomosis with the deep vessels, start on the periphery of the lung, and course along as a fine network between the lung and the visceral pleura, ramifying in the interlobar fissures, and eventually finding their way to another set of glands, situated at the hilum, where some anastomosis with the deep system takes place.

There is also a fine network of lymphatic vessels more intimately connected with the larger bronchi, consisting of two networks—a submucous, beneath the mucous membrane, and the peribronchial outside the walls of the bronchi. The importance of the distribution of the lymphatics will be evident later.

The course of affairs in the *development of silicosis* is as follows: dust enters the lungs by the air passages, and may set up a transitory and mild degree of bronchitis. It has been shown repeatedly that it is only the smallest particles of siliceous dust, having a diameter of one to three microns (one to three twenty-five thousandths of an inch), which gain entrance to the alveoli of the lung and produce the disease. Those which do not reach the alveoli are probably caught by the mucous secretions of the air-tubes, and carried away by the ciliated epithelium and by expectoration. In the early stages of the disease, at any rate, they do not appear to cause any marked degree of bronchitis, nor any recognizable

peribronchial fibrosis. The finest dust-particles are taken up by certain phagocytes derived from the alveolar epithelium, and carried by them to the nearest lymphoid tissue, and hence into the lymphatic vessels, *i.e.* they are deposited first in and around the lymph-nodules, which lie, as we have seen, in close relation with the terminal bronchioles, and also immediately under the visceral pleura. (Plate VI, Fig. 1.)

Gye and Kettle consider that finely-divided silica, which probably gets converted into a colloidal form in the lungs, acts as a slow cell-poison, sufficiently slow in its action to promote a progressive local fibrosis, but in the absence of infection not sufficient to produce necrosis. (Plate VI, Figs. 2, 3, 4.)

Heffernan believes that silicosis is the result of a local physico-chemical action of hydrated silica on pulmonary tissue, the silica absorbing moisture from neighbouring cells and, in effect, "drying them up." The dust-cells are also probably disintegrated by the same process, the peculiar action of silica dust in the form of particles of from 0·5 to 5 microns depending on its solubility. The speed of development depends on the rapidity of formation of fresh silica hydrosol. Alkalies accelerate the formation (*e.g.* in silica scouring-powders), while carbon and clay retard it.

From the lymph-nodes some of the laden dust-cells are next carried along a further set of lymph-channels, and eventually many of them reach the tracheo-bronchial and paratracheal glands at the roots of the lungs, where further fibrotic changes tend to take place. As the dust-cells congregate, through repeated exposure of the patient, the lymph vessels tend to dilate and a certain amount of general lymph-stasis occurs. This stasis is probably only a transitory phenomenon, but one often sees evidence of it in

radiographs of early stages of the disease, showing as an increase in the number and extent of the linear "striations" radiating from the hilar areas, so that practically the whole of both lung-fields is occupied by what is sometimes referred to as a "fine general arborization." In a radiograph of a normal lung the linear striations should only be seen extending about two-thirds of the way across the lung-fields, the peripheral third appearing clear and free from any but very faint linear shadows only seen on a good skiagram.

Radiologically and pathologically it is convenient to divide the disease into three stages: early, intermediate and advanced uncomplicated silicosis; but this classification will probably have to be modified in the light of recent studies. It is found that the three stages to be described, while holding in many cases, do not always follow each other, the type of fibrosis and its progress depending on the type of dust-industry, time-factors and the intensity of the dust inhaled in a given period. As new occupational fibroses are studied classification has become more difficult. It will be convenient, however, to describe three stages which are frequently found, and to discuss their modifications later.

In the *first stage* the hilum shadows are accentuated, denser and more extensive than normal. The pulmonary striations are also accentuated and extend well out to the periphery of the lung. This appearance generally begins in the right lower zone, and later extends symmetrically over both lung-fields. (Plate VIII, Fig. 7.) This picture is probably due, in this disease, to the clogging of the lymphatics with laden dust-cells, and to a degree of dilatation of these vessels with some slowing up of the lymph-stream. There is no alteration

in diaphragmatic excursion. The appearance at this stage is not characteristic; a similar picture may be produced by acute or chronic bronchitis (Plate VIII, Fig. 8), passive congestion of the lungs from cardiac decompensation (Plate IX, Fig. 9), irritation from certain gases (Plate IX, Fig. 10), and more rarely by metastatic or primary mediastinal malignancy.

The *second stage* is characterized by mottling throughout the lungs, due to small fibrotic nodules varying in size from a pinhead to a pea. The shadows are not very dense, are rather fluffy in outline, and have been likened to a snowstorm. This process usually starts in the right lung near the hilum, but soon assumes a more or less generalized distribution. There may be slight restriction of diaphragmatic movement. During this stage the accentuation of the hilum shadows and of the linear lung-striations usually disappears.

Describing this transition from linear markings to nodular fibrotic infiltrations, Steuart says: "There is an apparent segmentation of linear markings which now present themselves as numerous small, discrete, but clearly-aggregated miliary tubercles. This change from reticulation to nodulation usually progresses uniformly throughout the entire lung, and, as the condition advances, the nodules grow in size but apparently decrease in number."

Radiographically, then, the picture at this stage is very similar to miliary tuberculosis, except that the miliary infiltrations are due to fibrotic nodules and not to the tubercles of tuberculosis. The transition of the picture from linear markings to nodule-formation is explained by the clogging of the lymph-channels with dust-cells, stagnation of the stream, and the supervention of fibrosis. The radiographic appearances

of the typical "second stage" of silicosis are shown in Plate X, Figs. 11, 12, and the macroscopic features in Plate VII, Fig. 5.

The condition has to be differentiated from miliary tuberculosis, acute (Plate XI, Fig. 13) or chronic (Plate XI, Fig. 14), broncho-pneumonia, carcinomatosis (Plate XII, Fig. 15), and some rare mycotic infections (sporotrichosis). A careful consideration of the history and clinical findings is of the utmost importance in coming to a conclusion.

In the *third stage* the fibrosis extends, and the isolated nodules enlarge and coalesce into coarser nodules and patches of fibrosis. There is probably a leakage of dust-cells through the walls of the distended and blocked lymphatics, causing a more diffuse fibrosis in the interstitial lung tissues. This is possibly the cause of a re-appearance in this stage of the accentuated linear markings and dense hilum shadows seen in the radiograph. It is suggested by some observers that, with the blocking of the lymphatics of the lung, the lymph-flow is dammed back, and tends to find its way to the hilum by the pleural route. This gives rise in extreme cases to thickening of the pleura, adhesions and occasionally pleural effusion. Pleural involvement usually results in restriction of the excursion of the diaphragm. In rapidly-progressing cases this interstitial and pleural involvement may occur early, and the nodular stage be masked or fail to develop. This has been observed in the case of granite-cutters.

The skiagrams in Plates XII, XIII, Figs. 16, 17, from cases in the "third stage," illustrate these points. In the radiographic differential diagnosis these appearances have to be distinguished from those found in chronic fibroid phthisis, fibrosis from chronic infections (Plate XIII, Fig. 18), chronic interstitial pneumonia of

FIG. 1.

Photomicrograph. Pigmented cell aggregates.

A longitudinal section of a terminal bronchiole and its vestibule is shown. Note the accumulation within the walls of these structures of very numerous "dust cells," amidst which a few fibroblasts appear.

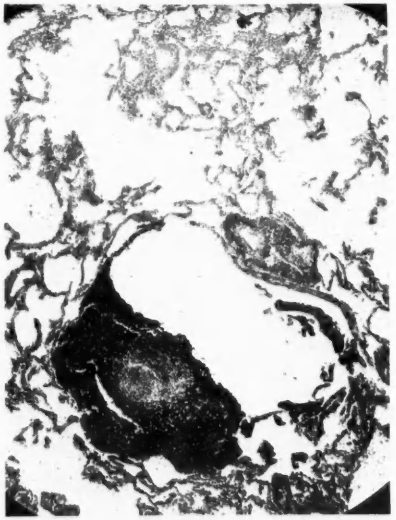

FIG. 2.

Photomicrograph. Commencing nodule formation.

A terminal bronchiole is shown with a related aggregate of pigmented "dust cells" in which a commencing nodular silicotic fibrosis is seen. The focus of fibrosis is still in the cellular stage.

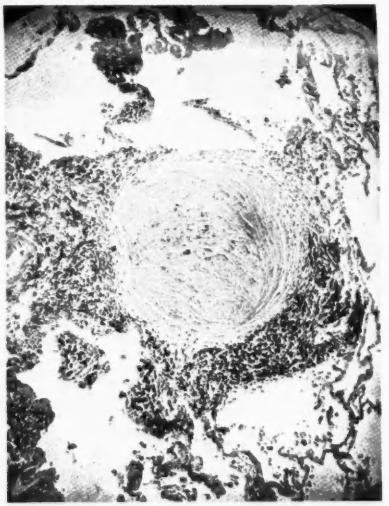

FIG. 3.

Photomicrograph. Fully formed single small "non-infective" silicotic nodule.

Note that the simple silicotic islet consists of a central area, composed of well-formed dense fibrous tissue with scanty cells and little pigment; most of the pigment is lying free. At the periphery of the islet the arrangement is that of concentric laminæ and the tissue is fibro-cellular. The islet is surrounded by an accumulation of pigmented "dust cells."

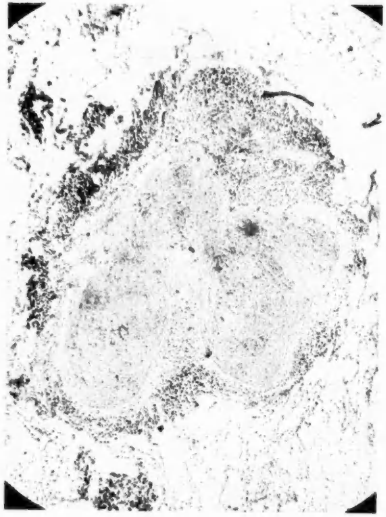

FIG. 4.

Photomicrograph. Composite "non-infective" silicotic nodule.

The section shows several small silicotic islets which were originally separate. The close proximity of their sites of origin has brought them into contact to form a "composite" structure. Note the sharp separation of the nodule from the surrounding lung tissue, which shows no significant change.

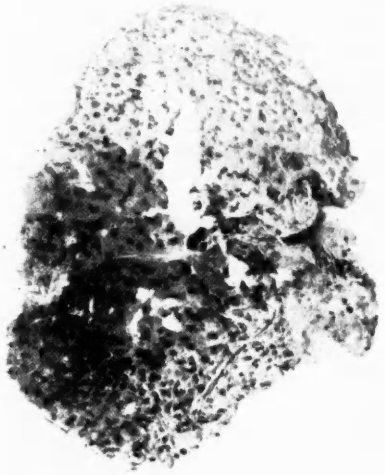

FIG. 5.

Macroscopic appearances in a moderate degree of silicosis of simple type.

Numerous discrete pigmented islets appear on the cut surface of the lung. They are mostly "small" and a considerable proportion are the seat of a palpable nodular fibrosis. The root glands are pigmented and fibrosed. A portion of pleura, with a few sub-pleural plaques, is visible on the right.

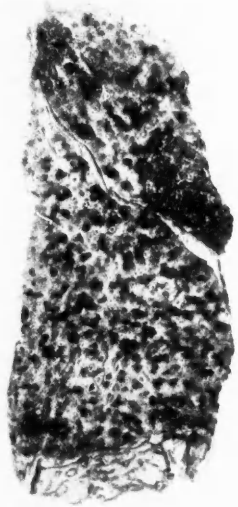

FIG. 6.

Macroscopic appearances in an advanced degree of silicosis of infective type. (Tuberculo-silicosis.)

Note the large irregularly-shaped nodules and in the upper lobe two areas of massive fibrosis of infective type. Material from the root glands and from the massive areas in this case was inoculated into guinea-pigs, and the animals in each case died from generalized tuberculosis.

composite structure. Note the sharp separation of the nodule from the surrounding lung tissue, which shows no significant change.

fibrosis. The root glands are pigmented and fibrosed. A portion of pleura, with a few sub-pleural plaques, is visible on the right.

this case was inoculated into guinea-pigs, and the animals in each case died from generalized tuberculosis.

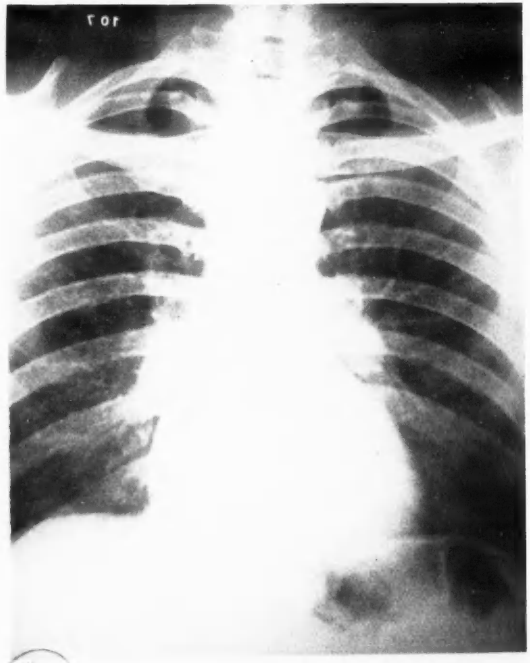

FIG. 7.
Silicosis. Early stage.

FIG. 8.
Bronchitis and emphysema.

PLATE VIII.

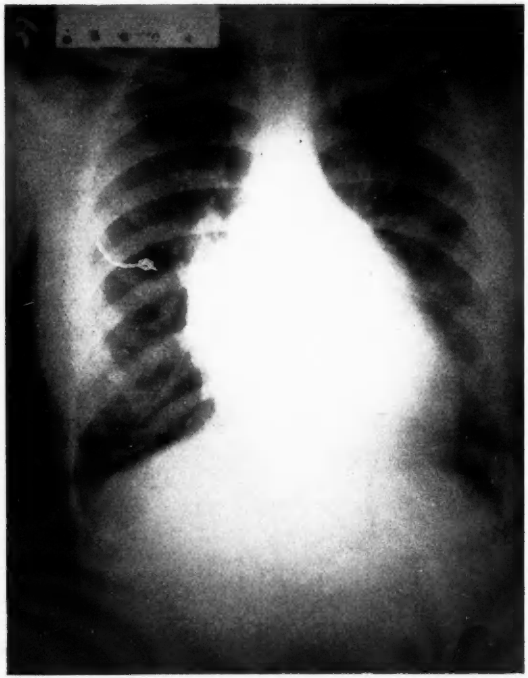

FIG. 9.
Passive congestion of lungs in heart disease.

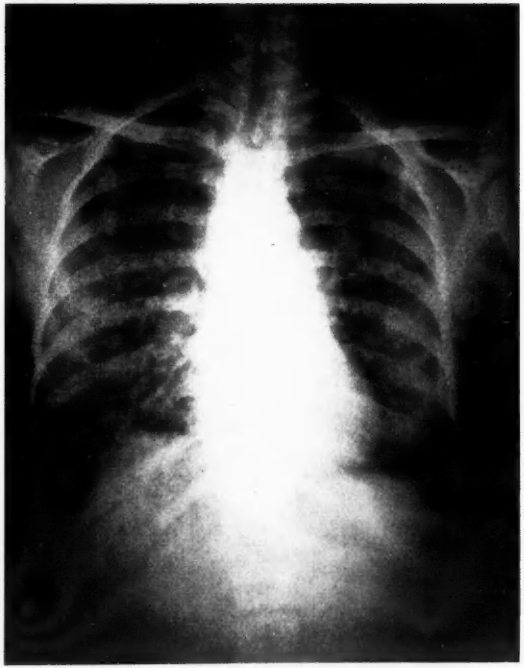

FIG. 10.
Fibrosis following gas-inhalation.

FIG. 9.
Passive congestion of lungs in heart disease.

FIG. 10.
Fibrosis following gas-inhalation.

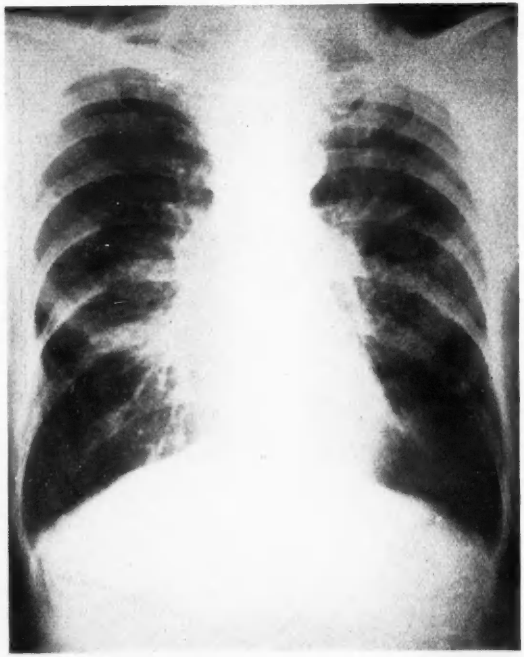

FIG. 11.
Silicosis. Commencing second stage.

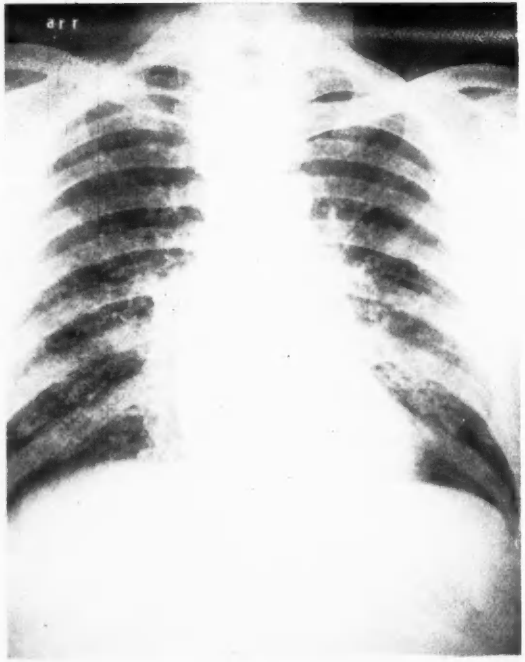

FIG. 12.
Silicosis. Second (nodular) stage.

PLATE X.

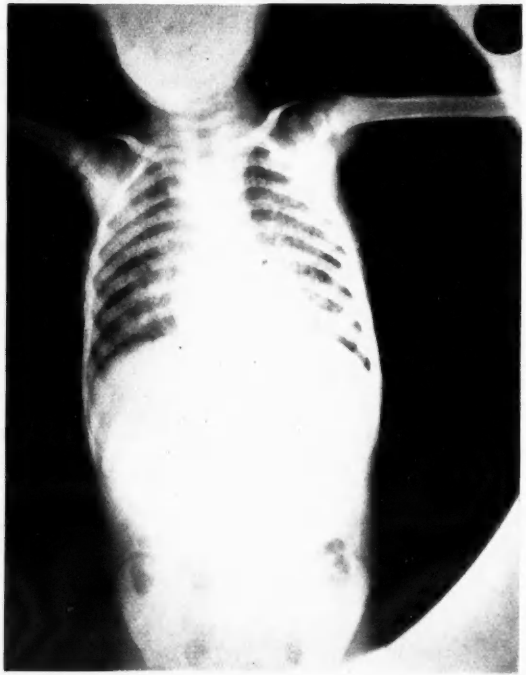

FIG. 13.
Acute miliary tuberculosis.

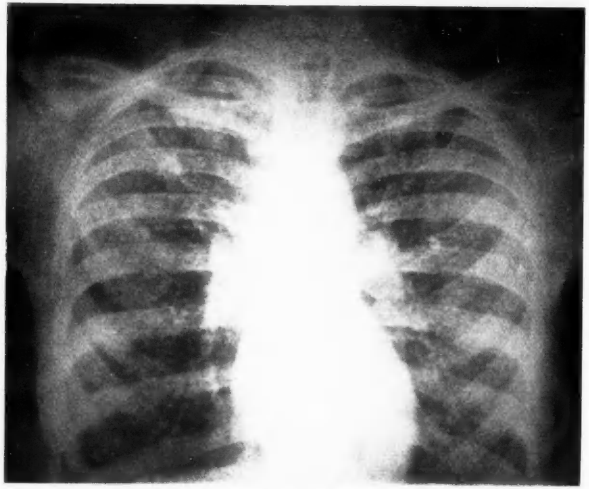

FIG. 14.
Chronic miliary tuberculosis.

PLATE XI.

Fig. 13.
Acute miliary tuberculosis.

Fig. 14.
Chronic miliary tuberculosis.

Fig. 15.
Carcinomatosis with effusion.

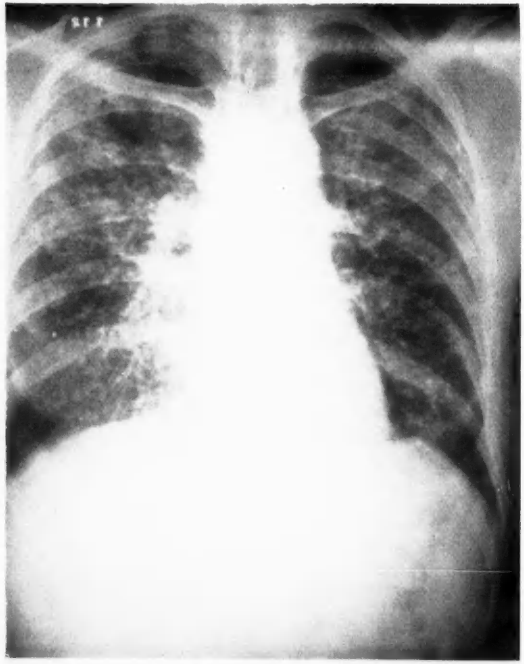

Fig. 16.
Silicosis. Advanced (third) stage.

PLATE XII.

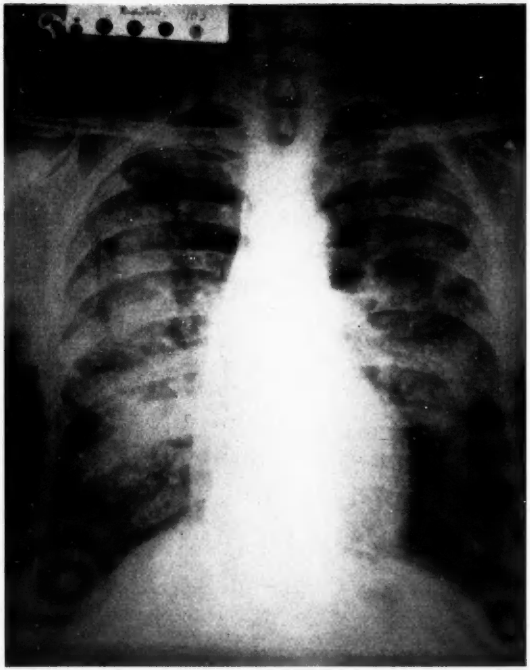

FIG. 17.
Silicosis. Advanced stage; marked pleurisy.

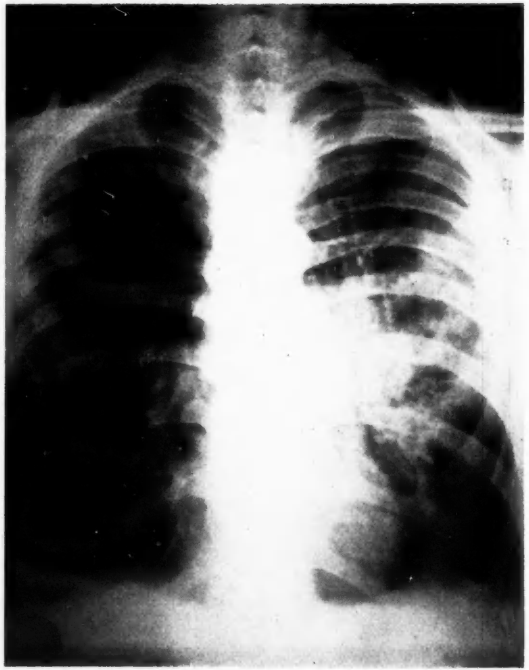

FIG. 18.
Fibrosis from Chronic Infection (non-Tuberculous).

FIG. 17.

Silicosis. Advanced stage; marked pleurisy.

FIG. 18.

Fibrosis from Chronic Infection (non-Tuberculous).

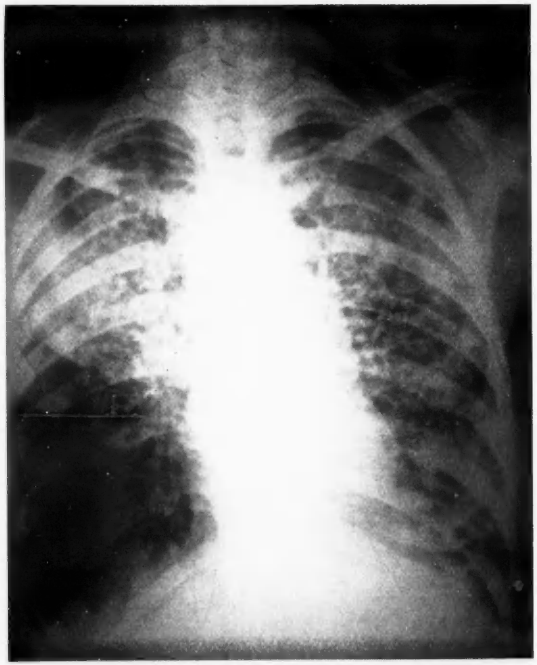

FIG. 19.

Multiple deposits of secondary sarcoma.

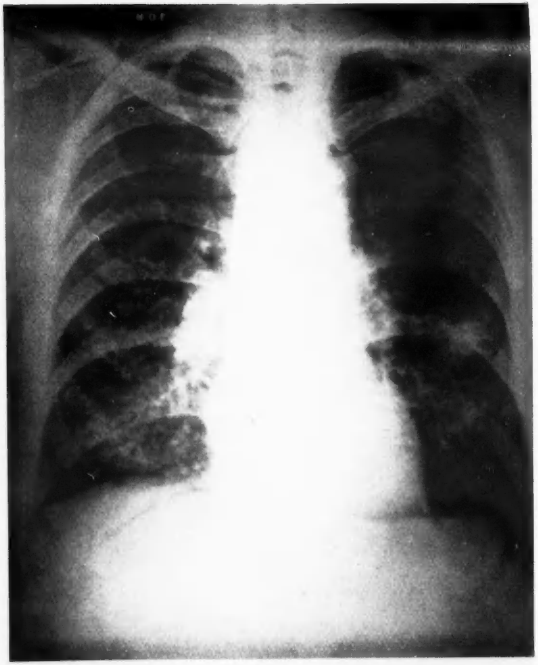

FIG. 20.

Silicosis. Late (third) stage. Infective focus in left middle zone.

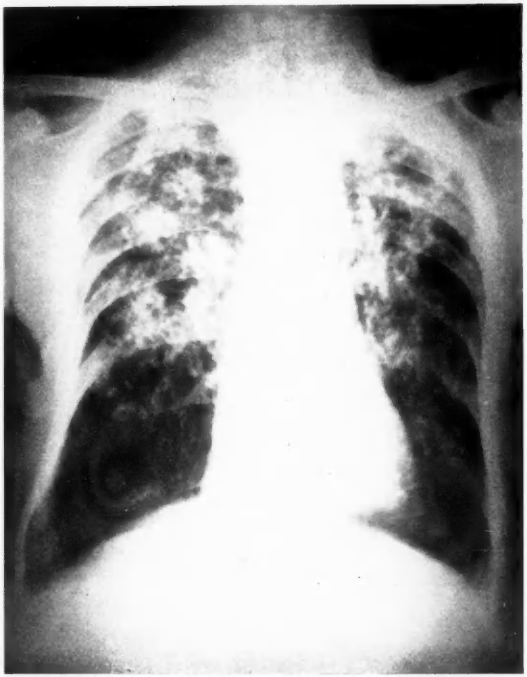

FIG. 21.
Tuberculo-silicosis. Cavity at left apex.

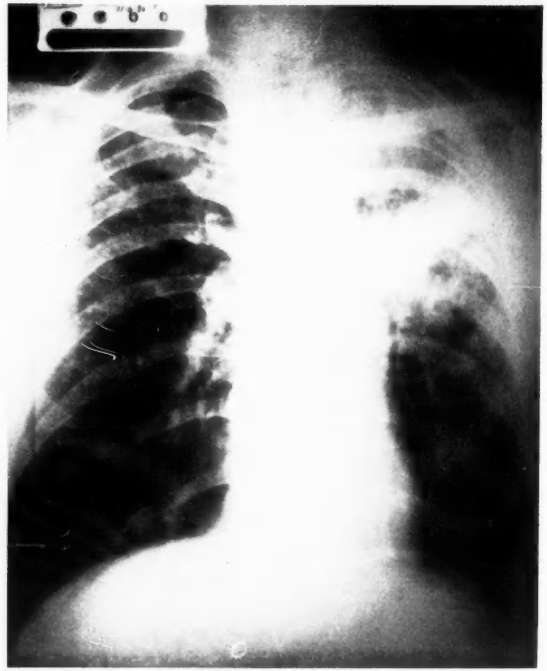

FIG. 22.
Tuberculo-silicosis.

PLATE XV.

pyogenic origin, tuberculous broncho-pneumonia, and multiple secondary deposits of new growth. Plate XIV, Fig. 19, is from a case of sarcomatosis. The question of the symmetry of the lesions and a careful consideration of the history, etc., will generally decide the diagnosis.

In cases of simple, uncomplicated silicosis the fibrotic changes may take anything from two to thirty years to develop, depending on the percentage of silica in the dust inhaled and on its intensity in a given period. Some early cases in the first or commencing second stage were observed recently in America to show actual regression of the disease during several months' enforced idleness during a strike. In other words, there seems to be a stage, in the absence of infection, where the pathological changes may actually regress, and a cure with little or no fibrosis may result. But this has rarely happened in the past, although with increasing knowledge preventive measures and early routine examinations we may be able to catch cases early enough before irreparable damage has been done.

These latter observations on the regression of the radiographical features of the early stages of the disease tend to support the foregoing interpretation of the shadows seen, namely, that the increase of linear radiations and the earliest stages of nodule formation are due to lymphatic congestion with dust-cells rather than to actual fibrosis. If the inhalation has not been excessive in point either of time or of intensity, such dust-cells can be eventually got rid of by normal physiological processes. Fibrosis, however, once established, leaves permanent scars, and the degree of disability will depend on the amount of the "physiological reserve" of the lungs which has been

Fig. 22. Tuberculo-silicosis.

Fig. 21. Tuberculo-silicosis. Cavity at left apex.

left intact, assuming that no super-added infection has complicated the picture. That the intensity of silica dust inhaled in a given period is as important as the time-factor is illustrated by four cases which were recently reported. In spite of relatively short exposures, sufficient dust was deposited in the lungs to set up a progressive fibrosis, which only produced symptoms many years after; *e.g.* one developed symptoms twenty-three years after exposure of only four months; another developed symptoms ten years after exposure of two years.

The main *symptom* generally complained of is an increasing dyspnœa on exertion with occasionally some unproductive nocturnal cough.

The *physical signs* may be few and indefinite, being mainly those of a degree of emphysema, with restricted air-entry. These cases may succumb eventually to right heart failure, due to the emphysema (rather than the fibrosis) interfering with the pulmonary circulation.

The degree of disability may not be great until the third stage is reached, but if infective complications are super-added, even in the early stages of the disease, the story is a very different one. This brings me to the point where we must discuss the disease *when complicated by infection*, often referred to by the South African authorities by the term "infective silicosis."

The infection is nearly always tuberculous in nature, either due to the flaring up of an old focus already present in the lung, a focus which has lain dormant but is potentially active, or to the inhalation of fresh bacilli, which find a happy hunting-ground among the already partially-poisoned cells of a silicotic lung.

Subjects of advanced silicosis more often contract tuberculosis than pneumonia, but the latter is more often found as a complication in earlier cases, where the inhalations are copious and the fibrosis slight (Belt). Two new pathological processes now make their appearance, namely focal necrosis and a tendency to excessive fibrous tissue formation. It is probable that the old text-books were wrong, and that necrosis never takes place in silicotic nodules in the absence of super-added infection. With regard to the excessive fibrous tissue formation, it is generally found that tuberculosis and silicosis together produce greater fibrosis than either alone. The fact that the silicotic lung is peculiarly prone to invasion by the tubercle bacillus has long been known, but is none the less interesting as a pathological problem. There is a mechanical factor at work, namely that the lymphatic drainage system of the lungs is already considerably impaired. In addition, many of the cells are poisoned by the soluble silica, and thus the cellular defensive mechanism of the lung is largely thrown out of action. Further, it is possible that the "adsorptive" powers of colloidal silica referred to previously may bind the complement of human serum, and so antibodies are prevented from attacking the bacilli.

Table of a Series of Cases Examined (1929–31).

Total number examined for possible silicosis	124
Total number found to have definite silicosis	91

Of these 91 cases, positive for silicosis, 71·6 per cent. were cases uncomplicated by infection; 28·4 per cent. were infective cases, probably tuberculous.

Of the uncomplicated cases of silicosis, there were :—

Stage 1 (early cases)	11
Stage 2	26
Stage 3	28

Of the infective cases over 90 per cent. were in the advanced (third) stage.

Of the 33 remaining cases of the series which were negative for silicosis, 12 had chronic fibroid phthisis, and 21 had a slight degree of chronic bronchitis, emphysema or old pleurisy.

The interesting point brought out by this analysis is the fact that infective complications were commonest in those cases which were classified as belonging to the third or late stage of the disease. It is possible that the radiographic appearances hitherto described as typical of this late stage may be, after all, but modifications of the so-called second stage, due to infection, and that once the typical, discrete, nodular and linear shadows of uncomplicated silicosis begin to become blurred and confluent and massive shadows appear, then we must deduce that infection is already present, and that if followed up and re-examined at intervals these cases will soon show signs of increasing consolidation, caseation and necrosis, with cavity-formation, or a rapidly-progressing fibrosis such as is never seen in silicosis or tuberculosis alone.

In pre-war days dust conditions were very much worse than now, fibrosis was relatively more marked in silicotic lungs, and cases of advanced silicosis were often seen which showed no clinical evidence of infection. These patients died from heart-failure, with ascites, œdema and marked dyspnœa with cyanosis. Such cases are not so often seen nowadays,

but cases in the late stage who have escaped active breakdown and are still classed as cases of simple silicosis nevertheless show radiographic and often clinical signs of a latent infection. Ultimately the infective element tends to gain the upper hand, and the condition becomes one of definite "tuberculosis with silicosis."

The radiographic recognition of "tuberculosis with silicosis" is sometimes very difficult, but serial studies and the observation of rapid changes will make the diagnosis more certain. The radiograph should show the characteristic signs of both diseases. In addition to the generalized and symmetrically distributed signs of silicosis in one or other of its stages, irregular blurring of the discrete nodular shadows is seen, together with extensive local opacities representing tuberculous consolidation, the latter often showing translucent areas of cavitation within them and showing an asymmetrical distribution. (Plates XIV, XV, Figs. 20, 21, 22 and Plate VII, Fig. 6.)

The clinical history and findings must be carefully considered in connection with the radiographic findings in these cases, since these appearances can be closely simulated by those found in a case of chronic fibroid phthisis with a recent bronchogenic spread. The latter may produce diffusely-scattered discrete lesions which mimic the coarse mottling of the later stages of silicosis, but they are usually less generalized and less well defined. First-class skiagrams are essential.

In a few cases the diagnosis must remain presumptive only, but here serial radiographs at suitable intervals are of great value in coming to a conclusion.

An interesting point, which needs elucidation, arises from a study of cases of silicosis, namely the

fact that bronchiectasis is rarely found in association with the disease when uncomplicated by infection; and even in those cases where tuberculosis has supervened bronchial dilatation appears to be rare, when one considers how frequently it is found in other cases of lung-fibrosis following an acute or a chronic infection.

The explanation probably lies (1) in the peculiar character and distribution of the fibrosis in silicosis, and (2) in the fact that pathological changes are relatively slight in the walls of the air-tubes as a result of the dust-inhalation, whereas, as a sequel to infections, degenerative changes in the bronchi may be marked.

There is not time here for more than a few remarks about the allied condition of lung-fibrosis known as *asbestosis*. The various forms of commercial asbestos are essentially a mixture, in varying proportions, of hydrated silicates of magnesium, aluminium, and iron, the former preponderating. In its main clinical features asbestosis closely resembles silicosis, but it differs from it (1) in the mode of distribution of the fibrous tissue in the lungs, this appearing more in the basal areas and in the interstitial tissues of the lung; (2) in its rather more rapid development; (3) in its radiological features, the fibrosis beginning to show itself in the basal areas as an increase of the linear radiations, and a diffuse blurring of detail, rather than the nodular formation seen in silicosis: the pleura also shows earlier signs of involvement, diaphragmatic restriction of movement appears early, and the lung-fields lack contrast and assume a " veiled " appearance, which has been likened to that seen on looking through ground-glass; (4) in a lessened susceptibility to the supervention of pulmonary tuberculosis compared with silicosis (though these cases more frequently

contract pneumonia as a complication); and (5) in the early appearance in the sputum, and in the lungs themselves, of the peculiar "asbestos bodies," which are yellowish-brown, of elongated bead-like form, often with bulbous ends.

SUMMARY.

The typical results of the inhalation of finely-divided silica dust over long periods of time, or in strong concentrations, is first to produce what may be called a chronic lymphatic congestion, with possibly some bronchiolitis, a stage from which recovery may be expected with little or no disability or fibrosis; then to produce the simple nodular "dust-fibrosis" which is silicosis; and, finally, in most cases to lead on to a condition of "infective silicosis" and to a true "dust-phthisis."

Individual cases show many variations in character and mode of development, depending on the factors of time, intensity and infection, and on the nature of the dust inhaled.

Pathological changes in the lymph-nodes previously effected in early life by tubercle and other agencies, together with the initial physique and the degree of inherited or acquired resistance to infection, all have an influence on the rapidity of onset and progression of the disease.

In conclusion, I am indebted to Dr. Sutherland of the Home Office, and to Drs. Keating and McVittie of the Bristol Silicosis Board, for permission to show some of the skiagrams which I have made for the Board; also to Dr. Peter Kerley for the loan of one or two additional skiagrams, and to the South African Mine Medical Officers' Association for much valuable

material, including the beautiful pathological plates which you have seen.

The fine radiographic details illustrating the pathological changes in the lungs, particularly in the early stages of the disease, have unfortunately been to a great extent lost in the process of reproduction of the original skiagrams.

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PLATE XVI.

FIG. 1.

FIG. 2.

Congenital "Lop-ear."

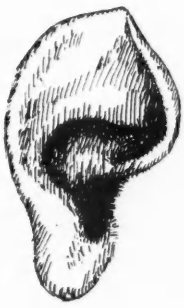

FIG. 3.
"Faun's Ear."

FIG. 4.
Double accessory auricle: note also the deformity of the pinna.

PLATE XVII.

FIG. 5.
Uvoid accessory auricle.

FIG. 6.
Congenital microtia.

FIG. 7.
Congenital microtia.

CONGENITAL ABNORMALITIES OF THE EXTERNAL EAR.

BY

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CONGENITAL deformities of the external ear are not very uncommon. They may consist merely of a departure from the usual shape sufficiently pronounced to attract attention; for example, absence of the lobule, which is said to be relatively common among the mentally defective and criminals, and on the Continent classified as a "stigma of degeneration." Rarer types are the "lop-ear," in which the pinna or its upper part hangs down over the meatus (Figs. 1 and 2); and the "faun's ear," in which the tubercle of the helix is erect and conspicuous. (Fig. 3.) The last variety represents a condition normal in fœtal life, and which, by its relation to certain simian ears, attracted the attention of Darwin. All these types tend to be symmetrical.

Errors of excess produce the most frequent deformity, the accessory auricle. The common type is a simple tubercle situated immediately in front of the tragus. It tends to run in families, to affect boys, and is often unilateral. I have seen an example in which such a deformity occurred on the right side in father, son and grandson. Much more rarely one finds two accessory auricles on the same side, as shown in

Fig. 4. The brother of this little boy had a single accessory auricle on the corresponding side. A rare form of accessory auricle is the pedunculated uvoid lobule, found below the ear on the side of the neck. (Fig. 5.) It resembles the extra lobule normal in goats, and is interesting from its frequent representation in classical statues of satyrs. These lobules are commonly bilateral.

Errors of defect are less common. The pinna may be merely small, and when this is unilateral a curious effect is produced, as in the celebrated example from Devizes. In the more extreme cases of congenital microtia the external ear is represented merely by one or two small protuberances; while a shallow dimple indicates the position of the meatus. (Figs. 6, 7.) The bony meatus in these cases is often absent, and the middle ear may participate in the abnormality; not seldom the labyrinth and even the eighth nerve show defects of development. For this reason it is generally useless to attempt surgical restoration of the meatus, though there is enough of the middle-ear tract to give rise to mastoiditis. Rarely such a condition is bilateral, and causes deaf-mutism.

An aural fistula results from imperfect closure of the first visceral cleft. Just within the opening of the meatus a small orifice opens on the floor, and may communicate with a branchial cyst of some volume overlying the posterior part of the parotid; sometimes attention is attracted by the discovery of mucoid discharge from the ear. Surgical removal is quite feasible. A still more rare condition is a persistent cleft, running down from the ear between tragus and antitragus into the side of the neck.

HÆMATURIA IN THE PRE-ERUPTIVE STAGE OF MEASLES.

BY

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THE occurrence of a copious hæmaturia during the prodromal stage of measles is sufficiently rare to be recorded.

On the 1st January, 1931, a boy, six years of age, became ill with sneezing, indefinite malaise and slight backache. There had been no previous illness. On the evening of the second day he passed, per urethram, without pain, what was thought to be pure blood. I saw him at 8.30 p.m. and found him in bed with a temperature of 101·4° F. and a pulse-rate of 112. The pharynx and nose were congested, but the retinæ, skin, lungs, heart, abdomen and limbs revealed nothing abnormal. The spleen was not felt, and there was no trace of œdema. About 10 fluid ounces of bright red, hæmorrhagic urine lay in a receptacle by the bedside, and on microscopical examination, without centrifugalizing, this was found to be full of erythrocytes.

On the 3rd January the temperature sank to 98° F. The urine was still full of blood. During the 4th and 5th January the temperature rose steadily to 101° F.; a cough developed, and there was slight photophobia, but there was much less hæmaturia. After my visit on the 5th January, the fourth day of the illness, the parent observed a rash appear on the forehead and spread rapidly downward, so that by the following afternoon, when I saw the child again, the face, trunk and limbs were covered by a morbilliform rash. Koplik's spots were not found, but they had had time to fade during the eighteen hours the rash had been out. The rash was not unduly hæmorrhagic and was typical. The rapid development of respiratory signs confirmed the diagnosis of measles. By the 11th January the exanthem had faded, leaving the characteristic faint brown staining and all trace of blood had gone from the urine. A mild broncho-pneumonia kept up the temperature, but convalescence was established by the 20th January. At no time was the spleen felt, and there were no other hæmorrhages.

About ten days after the onset a younger brother, aged eighteen months, developed typical measles. There were numerous other cases in the neighbourhood.

On the 3rd February, 1931, Dr. J. A. Birrell, Hon. Physician to the Bristol Royal Hospital for Sick Children and Women, was kind enough to take the boy into hospital for investigation. He reported that radiographs showed no trace of calculus; repeated urinary examinations could detect neither albumen nor blood; and the urea concentration test gave percentages of 2·2 and 2·8. The cardio-vascular system was normal.

On the 10th December, 1931, I found that the boy had been in good health throughout the year. The urine and its deposit showed no abnormality.

It is evident that this was not a case of hæmorrhagic measles, as the hæmorrhage was confined to one system only, and occurred only in the prodromal period. The hospital investigation and the absence of other clinical signs of nephritis and calculus, at the time of the illness, rule out these conditions as a cause of the hæmaturia. A careful search through the available literature[1] failed to discover a similar case. John Ruhräh notes: "Epistaxis and other hæmorrhages may occur, especially on the third or fourth day"[2] (of the prodromal period); whereas Dawson Williams, describing the same period, says: "In some epidemics, epistaxis has been observed in a large proportion of cases, occasionally to a serious extent."[3] I think that the hæmaturia in this case must be a hæmorrhage of the same type as the epistaxis noted by Ruhräh and Williams, that is, either toxic or congestive in origin.

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CYANOSIS IN THE FATAL BRONCHO-PNEUMONIA OF INFLUENZA.*

BY

FRANK E. FLETCHER.

AFTER an historical survey of the causation and pathology of influenzal broncho-pneumonia, the essay is devoted to a consideration of the causes of cyanosis in that disease, and proceeds:

In the bad cases of influenzal broncho-pneumonia one of the symptoms is a peculiar blue or violet cyanosis. J. S. Haldane[1] observes: "The cyanosis was attributed to the broncho-pneumonia, but against this explanation was the fact that the administration of oxygen failed to abolish or decrease the cyanosis, whereas the cyanosis usually accompanying broncho-pneumonia can be relieved or abolished easily by adding some oxygen to the inspired air." He suggested, on the evidence that he gave, that the hæmoglobin was in the form of methæmoglobin, and that this was converted post-mortem to nitric-oxide hæmoglobin. Sadie[2] showed this to be improbable, inasmuch as he demonstrated that, although there was a marked oxygen desaturation of arterial blood, it was not due either to methæmoglobin or sulphæmoglobin.

The gases in the arterial blood in several cases have been estimated, and it was seen that there was

* Part of an Essay for which a Martyn Memorial Pathological Scholarship was awarded, 1931.

a definite oxygen desaturation of the arterial blood. It would seem probable that the factors producing this great oxygen want were of such a nature as to allow large quantities of venous blood to return to the left side of the heart and so pollute the general circulation. It was seen that the carbon-dioxide content of the arterial blood in these cases was not diminished, not even to any great extent with the administration of oxygen. It has been suggested that this indicates that there was a condition present which prevented carbon-dioxide from being removed from a considerable number of alveoli in spite of increased ventilation. In other words, there were parts of the lung through which the circulation continued more or less freely, but the alveoli of these areas were potentially cut off from being ventilated under existing conditions. That the isolation of these areas may sometimes be incomplete was indicated by the fact that, if the partial pressure of oxygen in the inspired air was sufficiently high, the air in such alveoli was eventually enriched and supplied the blood with oxygen. It was noticed, too, that where the arterial oxygen saturation and carbon-dioxide content were estimated, there was parallelism between the oxygen desaturation and carbon-dioxide content of the arterial blood; the greater the former the greater the latter.

As the disease progressed there was a decline in the carbon-dioxide combining powers of the blood which was coincident with definite signs of marked consolidation of large areas of lung tissue. This would produce more or less complete cessation of circulation through these areas, and the venous blood returning to the general circulation would be less abundant.

The conspicuous change in the carbon-dioxide

dissociation curve is that there was a very high curve in the early stages, and as the disease advanced there was a progressive decline in the carbon-dioxide combining power of the blood, which never reached the normal curve. This has been explained by the occurrence, in the first instance, of a pronounced gaseous acidosis, which has been more or less compensated for by an increase in the alkaline reserve. It has also been determined that, whatever the oxygen saturation of the arterial blood in this disease, the dissociation curve is almost normal. It therefore seems clear that the oxygen want is directly the result of interference with the respiratory function in the lungs. It has been suggested that two mechanisms might lead to such interference. Firstly, areas of the lobules of the lung have been rendered non-functional by the closure of bronchioles without pronounced damage to the alveoli, and here the circulation will continue to a certain extent. The partial pressure of oxygen here, in the cut-off alveoli, will be rapidly reduced, while the carbon dioxide content will not be increased appreciably beyond an equilibrium with that of the venous blood. Hence numerous small areas of lung will be non-functional, although a certain amount of circulation is still possible. For instance, if a given amount of blood is circulating in a lung in which 50 per cent. is not working yet allows circulation, the blood only takes up 50 per cent. of its normal oxygen; therefore, venous blood will be returned to pollute the mixed blood.

The other mechanism leading to oxygen want is that of rapid and shallow breathing, which increases the relative dead space and, in addition, must give uneven ventilation of the pulmonary alveoli.

The cyanosis of carbon-dioxide retention is

plum-coloured. In rapid and shallow breathing there would be expected a deficiency of carbon-dioxide in the arterial blood proportional to the oxygen want, and this would lead to a pale greyish cyanosis; while the leaden grey to heliotrope type of cyanosis, met with in influenzal broncho-pneumonia, suggests a combination of the two.

In the following three cases of influenzal broncho-pneumonia the blood, withdrawn post-mortem, was examined spectroscopically.

Case 1. — Housewife, aged 51.

Habitat.—Unhealthy basement flat in healthy neighbourhood.

History.—Symptoms of influenza for about seven days. Then signs of explosive crepitation over base of left lung, and in patches over right lung. Next day dirty azure-blue cyanosis. Oxygen administered with very little improvement. Patient died on tenth day from onset of fever.

Post-mortem. — A well-nourished individual having at least two inches of subcutaneous fat. The lips still showed signs of cyanosis. The heart was enlarged and very fatty, weighing 22 oz., while there was fatty infiltration deeply into the muscle walls. A small quantity of blood was removed for examination. Over the whole of the posterior portion of the right lung and over the lower lobe were recent adhesions. Recent adhesions were also present posteriorly over the left lung. No definite areas of depression were seen, but the right lung felt soft and fleshy, with small areas which were more resistant, but the lung was still crepitant. On section it was seen to be the seat of hæmorrhagic œdema, and small areas of darker colour could be seen. Muco-purulent secretion was squeezed out of the bronchioles. Smears were made from this. The left lung showed the same changes as the right, only not as markedly. The trachea and bronchi contained muco-purulent secretion and showed signs of a recent bronchitis. The tracheo-bronchial glands were normal. The smears showed all Gram-positive organisms; a few staphylococci, but a far greater amount of streptococci, in chains varying from five to eight cocci in length. There were also present a few cells undergoing degenerative changes; these were thought

to be desquamated epithelial cells, and in some less changed cells cocci resembling isolated streptococci were seen. The blood was examined with a spectroscope, and showed the oxyhæmoglobin bands. It was also easily reduced to hæmoglobin. There were no signs of either methæmoglobin or nitric-oxide-hæmoglobin.

Case 2.—Male tramp, aged 45.

Habitat.—Casual wards.

History. — Had had a "cold" for about ten days. Complained of feeling giddy and faint.

Sat down by side of road and died. Police constable who saw him immediately after death said his lips were dark blue.

Post-mortem.—An extremely emaciated subject. There were no fat reserves anywhere; the omentum was thin and transparent. There were signs of mesaortitis in the arch of the aorta and the orifices of both coronary arteries were partially occluded. Patchy fibrosis of heart muscle was present. There were recent pleural adhesions over the left lung, but not on the right side. The left lung presented the same appearances as the right lung of the preceding case. The right lung, although no definite areas of consolidation could be felt, appeared to be more airless and hæmorrhagic than usual. The trachea and bronchi showed the same changes as Case 1. The tracheo-bronchial glands were normal. Smears and blood were taken as before. Almost the only organisms present in the smears were medium length streptococci, the majority varying from five to ten cocci in length. A spectroscopic examination of the blood showed the bands of oxyhæmoglobin only; no methæmoglobin was detected.

Case 3.—Publican, aged 47.

Habitat.—Slum area.

History.—"Feverish cold" for about five days. Found dead in beer cellar. The doctor who saw him immediately after death said his lips and neck were "slatish blue in colour."

Post-mortem.—A well nourished man, still showing signs of cyanosis. The right heart was very congested, the mitral valves were considerably thickened and showed that there must have been some regurgitation of blood during life. A sample of blood was taken. Excepting that it was the right lung which was chiefly affected, the lungs were almost identical with the last case. The trachea and bronchi contained a fair

quantity of muco-purulent pus, and atrophy of the mucous membrane, the cartilaginous rings being very well marked. The tracheo-bronchial glands were slightly enlarged and soft. The smear from the exuding bronchioles showed numerous streptococci in chains of three to ten cocci in length; occasionally they seemed to take a diplococcal form while in the chains. Several were seen either in or on desquamated epithelial cells, but their staining was not affected. There were also a small number of diplococci present, of smaller size than the streptococci. The blood again did not show the presence of methæmoglobin, but in this case the spectrum in a more concentrated solution showed the lines of hæmoglobin. In reducing the oxyhæmoglobin with ammonium sulphide care had to be taken, as a temperature much below the stated one—that of 50° C.—was sufficient to convert methæmoglobin into oxyhæmoglobin, and eventually into hæmoglobin.

These observations support the view that the cyanosis is due to (1) much of the blood circulating round non-functioning alveoli; (2) uneven ventilation of the functioning alveoli, brought about by increase of dead space. Fatal results are liable to occur suddenly with an already diseased heart when there is congestion in the pulmonary circulation, accompanied, as it must be, by lack of oxygen supply to its tissues.

My thanks are due to the Acting Police Surgeon of Bath, Mr. E. Scott White, for allowing me to perform the autopsies and to use his materials and laboratory.

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Reviews of Books

Applied Physiology. By SAMSON WRIGHT, M.D., M.R.C.P. Fourth Edition. Pp. xxviii., 552. London: Oxford University Press. 1931. Price 18s.—The rapidity with which fresh editions of this work appear is ample testimony to its well-deserved popularity. In this, the fourth edition, the general format of the whole has remained, but it has been thoroughly revised and much new information added. By the simultaneous deletion of obsolete matter this addition has been accomplished without any increase in the size of the book. The fresh work thus incorporated includes the recent studies on the physiology of the sinus caroticus, a discussion on the various theories of biological oxidation, and the clinical features of hyperparathyroidism and hyperinsulinism, to mention only a few. The author thus maintains his intentions of illustrating the principles of physiology by reference to clinical observations on the human subject, and the whole work has been brought completely up to date from the point of view of both pure and applied physiology. This book is almost indispensable, not only to the physiology student, but also to the clinician anxious to keep abreast with recent advances in physiology as they affect clinical medicine.

Ideal Marriage. Pp. 323. Price 25s. **Sex Hostility in Marriage.** Pp. 296. Price 17s. **Fertility and Sterility in Marriage.** Pp. 448. Price 25s. A trilogy by T. H. VAN DE VELDE, M.D. (Haarlem). Illustrated. London: William Heinemann Ltd. 1931.—The author maintains that medical men in general stand much in need of instruction in the psychology and technique of married life. His first volume is a manual of practical erotics that certainly leaves nothing unsaid. Experienced lovers will admit the thoroughness and the insight displayed, but have probably nothing to learn here; for others detailed text-book instruction is not what is necessary. The psychological aspects are emphasized throughout, especially the importance of equal and reciprocal satisfaction. The second volume deals rather with the psychology of married life as a whole than of its more intense and intimate moments, though these are not left in the background. Considerable space is devoted to "Choice of a partner." Here again the accuracy of the author's observations might be easier to defend than the practical value of his advice.

The third volume is devoted to the much-discussed subject of birth control; factors promoting fertility receive consideration as well as procedure available for modifying them. Contraception is dealt with in some detail, ethics and psychology being again in the forefront; apart from operative measures the author has no suggestions for avoiding the real disadvantages of well-known methods. The trilogy as a whole deals with an aspect of normal human life that is of supreme importance to nearly every man and woman, and about which instructed opinion has hitherto been little accessible. The author is to be congratulated on the straightforward but sympathetic tone maintained throughout.

Practical Morbid Histology. By R. DONALDSON, M.D., F.R.C.S.E. Second Edition. Pp. ix., 487. 214 illustrations. London: William Heinemann Ltd. 1931. Price 42s.—This is a welcome and much-improved second edition of Professor Donaldson's excellent handbook, which has been thoroughly revised and rendered more complete by the addition of several new chapters. In the previous edition illustrations were purposely omitted; the value of the present one is enhanced beyond measure by the inclusion of over two hundred original drawings. In a book of this kind, which is intended for the use of students, good illustrations are indispensable and cannot be adequately replaced by the most lucid of verbal descriptions; the author is therefore to be congratulated on deciding—although apparently with some reluctance, judging from his prefatory note—for their inclusion in the present volume. The chapters are well arranged in the orthodox manner, and the subject-matter has been carefully chosen. Little space has been devoted to theoretical considerations, for which the student is referred to the larger standard text-books. Professor Donaldson's purpose is to provide a useful practical companion volume to these standard works, and in this he has succeeded admirably. The illustrations were drawn specially for this edition by the late Richard Muir, and, needless to say, are uniformly excellent, losing none of their value for being at times somewhat diagrammatic. The book should prove extremely useful to students during their professional course, as well as to those sitting for higher examinations.

A Manual of General Medical Practice. By W. STANLEY SYKES, M.B., M.R.C.S. Second Edition. Pp. xi., 213. London: H. K. Lewis & Co. Ltd. 1931. Price 7s. 6d.—This is a well-written book, and deals with the subject of general medical practice in a very clear and useful manner.

It is highly recommended to all those recently qualified, and should be very helpful to them in particular. The object of the book, as stated in the preface to the first edition, is very well carried out. As each chapter is complete in itself as dealing with the varied aspects of general practice work and all are good it is difficult to single out one from the other, but perhaps those on "The Diseases of General Practice," "Diagnosis," "Prognosis," and "Treatment" will be found most useful, while that on National Health Insurance work is very clear.

The Cardiac Cycle. By HARRINGTON SAINSBURY, O.B.E., M.D., F.R.C.P. Pp. 79. Bristol: John Wright & Sons Ltd. 1931. Price 5s.—The argument proposed in this essay is that the pulse wave is set in motion by the impact of the blood driven by the pressure of the ventricular systole against the under side of the aortic valves before these open; that the vibrations which this initiates are transmitted to the column of blood in the aorta and its branches; and that the wave thus propagated, by "milking" the lymph along the perivascular spaces is largely responsible for maintaining the lymphatic circulation. We trust that these views will receive the respect that is their author's due, as well as the criticism that his prefatory note invites.

Clinical Notes on Disorders of Childhood. By D. W. WINNICOTT, M.R.C.P. Pp. viii., 216. London: William Heinemann Ltd. 1931. Price 10s. 6d.—This book is one of the "Practitioner's Aid Series," and deserves the attention of every general practitioner. The author has selected a remarkable series of case-reports accompanied by a running commentary on such important subjects as the rheumatic heart, the significance of "growing pains," anxiety states, enuresis and convulsions. His enthusiasm makes the reader feel that it is, as Noël Coward would put it, an exciting thing to be a general practitioner, "who must be the pioneer in the study of minor disorders." Much of the family doctor's work is a "specialized form of friendship." The author stresses the connection between the development of symptoms with difficulties in the emotional development of the child. Not every reader will agree with the doctrines of therapeutic, even psycho-therapeutic nihilism put forward, but everyone will be stimulated by Dr. Winnicott's fresh approach to matters of the greatest importance to the general practitioner.

Radiology in Relation to Medical Jurisprudence. By S. GILBERT SCOTT, M.R.C.S. Pp. x., 65. 24 plates. London: Cassell & Co. Ltd. 1931. Price 7s. 6d.—This little handbook adequately fulfils his aims as set out by the author in the preface. It describes many of those skeletal variations and other conditions which are liable to make accurate diagnosis difficult, and which often arise as problems to be decided in connection with claims for compensation in the Law Courts. The book is designed both for the medical man who uses X-rays for diagnosis, to help him to avoid making mistakes and to present radiological evidence efficiently in the witness box, and also for the legal profession, to help it to understand the properties of X-rays and their use and limitations in diagnosis. It consists of four chapters, covering in all some 60 pages, together with twenty-four excellently-reproduced radiographs. One might question the correctness of the author's views on one or two points, *e.g.* his notes on Kümmel's disease (p. 33), his terminology in the titles of Plates 9 and 10, and his suggestion that a hæmatoma will calcify in six days to the extent shown in Plate 15. But on the whole the handbook is well done, and is filled with information condensed in a form which should prove valuable to many who have not the time to familiarize themselves with the many normal variations and slight evidences of early pathology described in more detailed works.

Fundamental Principles of Ray Therapy. By W. BEAUMONT, M.R.C.S. Pp. viii., 124. 37 illustrations. London: H. K. Lewis and Co. Ltd. 1931. Price 6s. An elementary text-book should be clear and strictly accurate. This little book, which is certainly very elementary but neither clear nor accurate, treats ray therapy (we are told in the preface) in an unusual way. The number of errors in it is simply appalling. Need more be said about it?

The Epidemiology and Control of Malaria in Palestine. By I. J. KLIGLER. Pp. xv., 240. Illustrated. Chicago: The University of Chicago Press. 1931. Price 22s. 6d.—This volume describes the control of malaria as carried out in certain districts of Palestine in the last nine years, and is of more than local interest, because it shows how the scientific study and control of the local mosquito carriers may be applied to other countries. Palestine has suffered terribly from the disease. Entire towns and villages have been wiped out by it, and the early Jewish colonies were planted in what

turned out to be some of the worst districts. The malaria has certain peculiarities : the breeding season of the anopheles is remarkably prolonged, and there are two malarial special seasons in the year, viz. May to August and October to December. Complete hibernation does not exist. Even in January a few mosquitoes previously infected do enter houses carrying sporozoites, and cause new attacks. Again, though there are eight kinds of mosquitoes present, only three or four are of importance, and Kligler rightly lays stress on the folly and waste of attacking those which rarely infect man. Indeed, since this book appeared Swellingrebel has on the same principle changed the whole method of control in South Africa, showing that only two species there are important, and great sums have been wasted in drainage works for want of knowledge as to the true vectors. *A. Elutus* does most of the damage in Palestine, and breeds in stagnant or sluggish waters, while *M. Sergenti* and *M. Superpictus* in swift streams in the summer are also serious. In the salt water of a few marshes and around the Dead Sea we get *M. Multicolor*, and finally *A. Bifurcatus*, having developed a bad habit of breeding in house cisterns in towns, is much more harmful here than it is in Italy, where it leads an outdoor life. Our author, too, has the credit of proving that while the limit of direct flight is one and a half miles here and elsewhere, the actual range of dispersion of infected insects may be two and a quarter, and just before winter as much as five or seven miles. This, of course, is a very important matter. His method of control was, after a careful survey of the population and of the mosquitoes on the spot and for a mile or two around, (1) to drain stagnant pools and to clear away weeds from the water-courses, (2) to destroy lârvæ by spraying every seven or ten days with Paris green or a crude oil mixture, and (3) to organize hunts for adult mosquitoes in houses and stables. The results were first rate, except in three districts where huge marshes could not be treated. It is remarkable that he got no success when he tried careful quinine administration to an entire population. This is a marked contrast to the good results reported in many countries, as collected by Sir Leonard Rogers (*Recent Advances in Tropical Medicine*). Kligler was himself much surprised at the failure, and though he allows that blackwater fever vanished and that the loss of working time was greatly reduced where quinine was given, he is quite certain that the incidence of the disease was not lessened. Certainly the difference is not explained by arguments that quinine is not a prophylactic though invaluable in treatment.

We can warmly recommend the book as a very able and painstaking contribution to the study of malaria. Every page shows the author's infinite capacity for taking trouble, every little detail is illustrated by elaborate maps, charts and tables of statistics. We may add that it is brightened by many charming little photographs of marsh scenes.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S. Third Edition. Pp. xx., 277. Illustrated. Bristol: John Wright & Sons Ltd. 1931. Price 21s.—We are not at all surprised that a third edition of this most excellent book has been called for in four years. Students, doctors and professional surgeons will all do well to read it. To master its contents is to go far towards becoming a good surgical diagnostician, and even to glance over its 318 illustrations, well chosen and well printed, is a quick and easy means of improving one's education. We are glad to see that the author calls attention to the value of trans-illumination in the diagnosis of swellings in the breast. There are a few misprints, *e.g.* in the title of Fig. 164.

A Pocket Atlas and Text-Book of the Fundus Oculi. By G. LINDSAY JOHNSON, M.D., F.R.C.S. Second Edition. Pp. ix., 215. Illustrated. London: Adlard & Son Ltd. 1931. Price 12s. 6d.—The twenty-seven beautiful plates of the fundus oculi, giving fifty-four illustrations of diseases of the fundus, are in every way excellent, and as each is combined with a very clear footnote, this book should prove a very useful guide to ocular conditions for the student and busy practitioner. These plates have an introduction in the letter-press, which, although not strictly dealing with the fundus, gives a very clear discussion of cataract, glaucoma, etc., and a concise description of methods of examination of the fundus and difficulties met with in certain cases.

Warwick and Tunstall's "First Aid" to the Injured and Sick. Thirteenth Edition. By F. C. NICHOLS, M.C., M.B., Ch.B. Pp. xii., 276. Illustrated. Bristol: John Wright & Sons Ltd. 1931. Price 2s. 6d.—Dr. Nichols is to be congratulated on the thirteenth edition of this well-known handbook on first aid. This book, which is intended for the advanced ambulance worker, amplifies the information given in the authorized text-book of the St. John Ambulance Association, and provides that additional instruction which will be so

useful to those who have to assist in the training of junior first-aid students. The many illustrations are well reproduced, and the tabulation of treatment should be of value. The new chapter on "Gas Warfare" seems somewhat pessimistic, but will be of interest to those serving in territorial medical units, while of the other additions the chapter on "Competition Work" will be appreciated by the many men and women who take part in these competitions. One can confidently recommend this book to all those ambulance students who wish to pursue the subject beyond the elementary stage.

Gould's Medical Dictionary. Third Edition. By GEORGE M. GOULD, A.M., M.D. Edited by R. J. E. SCOTT, M.A., B.C.L., M.D. Pp. xvi., 1,538. Illustrated. London: H. K. Lewis & Co. Ltd. 1931. Price 30s.—The third edition of this dictionary is well up to the high standard of previous issues. In addition to many thousands of words described, there are numerous useful tables of bacteria, operations, tests, etc. The editor calls attention to the simplification of orthography that is one of his principal aims, but he is inconsistent in the matter, frequently and apparently capriciously retaining the older spelling. Apart from this we have no criticism to offer, but the highest praise for the excellent production of this standard work of reference.

Editorial Notes

Colston Medical Research Fellowships.

THE Council of the Colston Research Society have decided to use the Medical Research Fund collected by Mr. Falk for founding one or more Colston Medical Research Fellowships of not less than £100 per annum. The regulations governing these Fellowships are as follows, viz.:—

1. That the Fellowships be awarded by the Council of the Colston Research Society.

2. That they shall be Research Fellowships primarily in clinical subjects, but other medical subjects are not excluded.

3. That they be open to any member of the medical profession in Bristol, preference being given to graduates or members of the teaching staff of the University. Holders must possess a medical qualification, and their proposed work shall have been approved by the Council of the Society.

4. That the work shall be carried out in the University of Bristol or in any of the clinical institutions associated with the University.

5. That the holder be required to present a report at the end of the year on his work.

6. That the Fellowships be renewable yearly at the discretion of the Council of the Society.

7. That the Fellowships need not necessarily be awarded in any given year.

8. Applications should be made to the Hon. Secretary of the Society, 12 Small Street, Bristol, on or before 30th April of each year.

* * * * *

Medical Dinner.

THE Annual Medical Dinner was held on Thursday, 10th December, at the Clifton Zoological Gardens, under the Chairmanship of Dr. Charles Corfield. It was attended by nearly one hundred and fifty guests, the majority of whom were past or present Bristol students, thereby giving the Dinner the character of a reunion for the Bristol Medical School. This was the idea with which the Dinner was originally founded in 1882.

The guest of the evening was Professor D. C.

Rayner, who holds the Chair of Obstetrics in the University of Bristol, and is an alumnus of the Bristol Medical School.

The speeches were admirably short and to the point, whilst the banqueting-room and the catering arrangements gave the greatest satisfaction. In the course of the evening the Dean (Professor Fawcett) announced that a Committee had been formed to arrange the Centenary Celebrations of the Bristol Medical School in 1933.

* * * * *

Edward Jenner's Books for the Medical Library.

DR. ACHESON of Berkeley recently presented to our Library four interesting books which had been the property of Jenner, three of them still showing his signature. The great collection of Jenner's relics which was on exhibition here some years ago having gone to London and the United States permanently, we are fortunate to get these few books from his library.

The first we take up is *Observations on Scrophulous Affections*, by Robert Hamilton, M.D., published in 1791, an abstract of which had been read by Lettsom before the Medical Society of London and praised by it. The author at that time was a rising man, who had spent some years in the Army, in successful private practice, but unfortunately he became blind and remained so for thirty years.

Next we find a presentation copy of J. Mason Cox's *Practical Observations on Insanity*, dated 1806, the second edition. Cox was a Bristol man who graduated at Leyden, practised like so many others in Unity Street, and carried on an asylum at Fishponds.

Another volume presented by the author to Jenner is Sir J. C. Hippisley's *Speech on the Petition of the Roman Catholics in Ireland* in 1810, with many documents on the subject. Hippisley, then living at Stoneaston, and member for Sudbury, belonged to a famous family connected with Bristol, and had himself done a good deal for the city. As a diplomat he had successfully negotiated the marriage of the Princess Royal of England with the Duke of Wurtemberg, and was made a baronet as a reward. He had also obtained relief from King George III for the poverty-stricken Cardinal York, the last of the Stuarts.

Finally, there is a fine volume with many illustrations, *A Complete Treatise of Electricity*, by Tiberius Cavallo, a naturalized Italian, Fellow of the Royal Society, and one of the ablest scientists of the time. Cavallo seems to have been especially noted for the delicacy and accuracy of his measurements in electricity and physics.

Meetings of Societies

Bristol Medcio-Chirurgical Society

SESSION 1930-31.

THE Annual General Meeting of the Society was held in the Physiological Lecture Theatre of the University on Wednesday, 14th October, 1931, at 8.30 p.m.

Professor D. C. Rayner was in the Chair, and ninety-two members were present.

The minutes of the previous meeting were read and confirmed.

Three ladies and five gentlemen were proposed for election to the membership of the Society.

The President, Professor D. C. Rayner, then resigned the Chair to his successor, Dr. J. R. Charles.

BRISTOL MEDICO-CHIRURGICAL SOCIETY.

INCOME AND EXPENDITURE ACCOUNT for the year ended 30th September, 1931.

INCOME.	£ s. d.	£ s. d.
Members' Subscriptions ..	300 15 6	
Do., outstanding for 1930–1931	55 2 6	
		355 18 0
Interest on Bank Deposits		3 16 9
Interest on £500 4 per cent. Consolidated Stock, less tax		15 10 0
		£375 4 9

EXPENDITURE.	£ s. d.	£ s. d.
Grant to *The Bristol Medico-Chirurgical Journal*		275 18 11
Librarian—Honorarium		15 0 0
Miscellaneous Expenses—		
Goodway–Refreshments	22 10 0	
Postages and Sundries	8 13 5	
Printing and Stationery	11 7 3	
Audit Fee	2 2 0	
Lantern and Cinematograph at Meetings ..	4 4 0	
University Marshal ..	1 0 0	
Lewis's Medical and Scientific Library—		
Subscription	4 4 0	
Postages ..	0 8 7	
	4 12 7	
		54 9 3
Balance, being excess of Income over Expenditure carried to Capital Account		29 16 7
		£375 4 9

BALANCE SHEET as at 30th September, 1931.

CAPITAL AND LIABILITIES.	£ s. d.	£ s. d.
Sundry Creditors		85 10 8
Capital as at 30th Sept., 1930	656 8 11	
Add Balance of Income and Expenditure Account as above ..	29 16 7	
		686 5 6
		£771 16 2

ASSETS.	£ s. d.	£ s. d.
Cash at Bank—		
Current Account	114 7 8	
Deposit Account	157 7 0	
		271 14 8
£500 4 per cent. Consolidated Stock at cost		437 9 0
Members' Subscriptions for 1930–1931 outstanding	55 2 6	
Roberts — Honorarium in advance for 1931–32 ..	7 10 0	
		62 12 6
		£771 16 2

Audited and found correct,
H. E. KEELER,
Chartered Accountant,
Auditor.

16–18 CLARE STREET, BRISTOL,
12*th October*, 1931.

Dr. Watson-Williams proposed a vote of thanks to Professor Rayner for his services during the past year ; this was seconded by Dr. Symes and carried with acclamation. Professor Rayner replied.

Dr. J. R. Charles then gave his inaugural address on **"Structure and Function."**

Mr. T. Carwardine proposed a vote of thanks to the President for his address ; this was seconded by Dr. Carleton and carried with acclamation.

The Hon. Secretary and Hon. Treasurer then gave their reports, and the Editor gave his report on the production and development of the *Journal*.

The President thanked them for their reports and for their work during the past year.

Professor Hey Groves was elected to the office of President-Elect, on the proposition of Professor Nixon, seconded by Dr. Kennedy.

Dr. Kyle proposed and Mr. N. Kemm seconded the election of Mr. H. L. Shepherd as Hon. Secretary. This was carried.

Mr. A. E. Iles was re-elected Hon. Treasurer on the proposal of Mr. Duncan Wood, seconded by Dr. R. C. Clarke.

Dr. Watson-Williams proposed that Dr. G. Parker be elected Hon. Medical Librarian ; this was seconded by Mr. A. E. Iles and carried.

The following gentlemen were elected to serve on the Committee, proposed by Dr. R. S. Statham and seconded by Dr. Ormerod :—

Dr. W. K. Wills.
Mr. H. Chitty.
Dr. H. G. Kyle.
Dr. Carey F. Coombs.
Mr. W. A. Jackman.
Mr. Duncan Wood.
Mr. A. R. Short.
Dr. W. P. Kennedy.
Dr. Elwin Harris (jun.).

Dr. G. Parker, Mr. A. R. Short and Dr. J. O. Symes were elected to serve on the University Sub-Committee for the Medical Library on the proposal of Dr. P. Phillips, seconded by Dr. E. G. Bradbeer.

The President then referred to the Annual Dinner, and suggested that, on account of the present national financial and political crisis, there should be no dinner this year. This was carried and, furthermore, that members should be asked to contribute to a fund, which should be forwarded as from the Society, half to the Medical Benevolent Fund and half to the Lord Mayor of Bristol's Relief Fund.

The second meeting of the session was held in the Physiological Lecture Theatre of the University on Wednesday, 11th November, 1931, at 8.30 p.m. Dr. J. R. CHARLES in the Chair.

The minutes of the previous meeting were read and confirmed.

Dr. A. D. FRASER showed a number of interesting pathological specimens.

Professor J. A. NIXON and Dr. G. B. BUSH read papers* on **"Silicosis,"** Dr. Bush illustrating his paper with a series of skiagrams and also with the epidiascope.

Dr. MAYES discussed the prognosis in cases of Silcosis, quoting a group of thirty in which only four were found to be living four years after his first examination.

Dr. CARLETON mentioned that catarrhal children also developed pulmonary fibrosis, but in them the condition leads on to bronchiectasis rather than tuberculosis. He inquired whether cases of silicosis also developed bronchiectasis or radial dilatation of the bronchioles. In his experience suction dyspnœa was common in fibrosis.

Dr. KENNEDY spoke of the effects of silica poisoning in the spleen, liver and stomach. He quoted a case of recovery from pulmonary silicosis.

Dr. ALEXANDER spoke of a patient, at present under his care, in whom silicosis was due to sand-blasting.

Dr. BERGIN congratulated Dr. Bush on the excellence of his skiagrams and stressed the value of radiology in the early diagnosis of silicosis.

The PRESIDENT said he considered that the expense to the workmen of examination by the Silicosis Board might act as a deterrent to justifiable claims, and spoke of the length of time the disease might exist before its presence was suspected.

Professor NIXON and Dr. BUSH replied.

The third meeting of the session was held in the Physiological Lecture Theatre of the University on Wednesday, 9th December, 1931, at 8.30 p.m. Dr. J. R. CHARLES in the Chair.

The minutes of the previous meeting were read and confirmed.

* See pages 253 and 259.

Dr. A. L. TAYLOR showed pathological specimens, and Dr. D. A. ALEXANDER showed a specimen of a lung from the case of silicosis which he had mentioned at the previous meeting.

Dr. R. G. GORDON and Dr. M. F. FORRESTER-BROWN showed a cinematograph film illustrating the **"Treatment of Anterior Poliomyelitis."**

A discussion on the film and its subject then followed.

We greatly regret that in the discussion on Dr. BRISTOWE's paper on **"The Sterilization of the Unfit,"** as reported in our last issue, a mistake was made in the reporting of Dr. A. N. P. Milner's remarks. What he actually said was :—

"Of recent years the Neo-Darwin theory appears to have fallen into disrepute ; even Shaw describes it as Unnatural Selection, while evidence is accumulating in favour of the theory of Creative Evolution. It is probably unwise to prophesy, but it may be that in future we shall have to revise our present attitude to Evolution and Mendelism, and possibly we shall find that inheritance occurs, not by structural transmission, but by the transmission of functional diatheses."

The Medical Library of the University of Bristol,

WITH WHICH IS INCORPORATED

The Library of the Bristol Medico-Chirurgical Society.

The following donations have been received since the publication of the list in September, 1931.

December, 1931.

Dr. D. R. Acheson (1)	4 volumes.
Blacker Library of Zoology (2)	1 volume.
Dental Board of the United Kingdom (3)	1 ,,
Glasgow Royal Maternity and Women's Hospital (4)	1 ,,
Prof. E. W. Hey Groves (5)..	1 ,,
London School of Hygiene and Tropical Medicine (6)	1 ,,
Michell Clarke Memorial Fund (7)	24 volumes.
Munro Smith Memorial Fund (8)	10 ,,
Royal College of Surgeons of England (9) ..	1 volume.

Unbound periodicals have also been received from Professor E. W. Hey Groves and Dr. Carey F. Coombs.

THE ONE HUNDRED AND THIRTY-EIGHTH LIST OF BOOKS.

The figures in round brackets refer to the figures after the names of the donors. The books to which no such figures are attached have either been bought from the Library Fund or received through the *Journal*.

Bailey, H. .. .. .. *Demonstrations of Physical Signs in Clinical Surgery* .. .. 3rd Ed. 1931

Bainbridge, F. A. .. *The Physiology of Muscular Exercise.* 3rd Ed. (8) 1931

Barritt, W. B., and A. T. *Simple Handbook of Dental Materia Medica and Therapeutics* .. .. .. 2nd Ed. 1923

Bayliss, W. M. .. .. *Principles of General Physiology* 4th Ed. (8) 1927

Blair, V. P., and Ivy, R. H. *Essentials of Oral Surgery* .. .. (7) 1926

Bowlby, A. A., and Andrewes, F. W. *Surgical Pathology and Morbid Anatomy* .. .. 8th Ed. (7) 1930

Boyd, M. F. .. .. *An Introduction to Malariology* .. .. (7) 1930

Boyd, W. .. .. .. *Pathology of Internal Disease* .. .. (7) 1931

Bulleid, A. .. .. *A Text-book of Bacteriology for Dental Students* 1926

Burchard, H. H., and Inglis, O. E. *Text-book of Dental Pathology and Therapeutics* .. .. (7) 1927

Castellani, A. .. .. *Climate and Acclimatization* .. .. .. (8) 1931

Cavallo, T. .. .. *Complete Treatise of Electricity* .. .. (1) 1777

Colyer, F. .. .. .. *Abnormal Conditions of the Teeth of Animals in Their Relationship to Similar Conditions in Man* .. .. (3) —

Cox, J. M. .. .. *Practical Observations on Insanity* 2nd Ed. (1) 1806

De Lee, J. B. .. .. *Principles and Practice of Obstetrics* 5th Ed. (7) 1930

Economo, C. von .. *Encephalitis Lethargica: its Sequel and Treatment* 1931

Fenton, W. J., and Burrell, L. S. T. *Diseases of the Chest* .. .. (7) —

Findlay, L. .. .. .. *Rheumatic Infection in Childhood* .. .. (7) 1931

Garrod, A. E. .. .. *Inborn Factors in Disease* .. .. .. (7) 1931

Hamilton, R. .. .. *Observations on Scrophulous Affections* .. (1) 1791

Harrison, L. W. .. *Diagnosis and Treatment of Venereal Diseases in General Practice* .. .. (7) 1931

Hart, B. .. .. .. *Psychology of Insanity* .. .. (7) 1930

Hippisley, Sir J., Bart. *Speech on the petition of the Roman Catholics in Ireland* .. .. 2nd Ed. (1) 1810

Hopewell-Ash, E. .. *An A.B.C. of Medical Hypnosis* .. .. .. —

Hornibrook E. A. .. *Restoration Exercises for Women* 1931

Hughlings-Jackson, J. *Selected Writings.* Vol. I. .. .. .. (7) 1931

Kligler, I. J. .. .. *Epidemiology and Control of Malaria in Palestine* 1930

Lewis, T. .. .. .. *Clinical Disorders of the Heart Beat* 6th Ed. (7) 1925

Lloyd, W. .. .. .. *Hay-fever, Hay-Asthma* 1931

Lyle, H. W., and De Souza, D. *Manual of Physiology* 3rd Ed. (8) 1930

McCarthy, L. .. .. *Histopathology of Skin Diseases* .. .. (7) 1931

Marshall, A. M., and Hurst, C. H. *A Junior Course of Practical Zoology* .. .. 11th Ed. (8) 1930

Masters, W. E. .. *The Alcohol Habit and its Treatment* .. 1931
Mathews, A. P. .. *Physiological Chemistry* .. .. 5th Ed. (8) 1930
Medical Evidence before the Royal Commission on Licensing, 1930 .. 1931
Mennell, J... .. .. *Backache* .. .. (7) 1931
Nattrass, F. J. .. .. *The Commoner Nervous Diseases* 1931
Newth, G. S. .. .. *A Text-book of Inorganic Chemistry.* New and Enlarged Ed. 1926
Plimmer, R. H. A. .. *Practical Organic and Bio-Chemistry.* New Ed. (8) 1926
Poynton, F. J., and Schlesinger, B. *Recent Advances in the Study of Rheumatism* .. .. (7) 1931
Pryde, J. .. .. .. *Recent Advances in Bio-Chemistry* 3rd Ed. (8) 1931
Singer, C. .. .. .. *A Short History of Biology* .. .. .. (8) 1931
Smith, S., and Glaister, J. *Recent Advances in Forensic Medicine* (7) 1931
Stewart, A. W. .. *Recent Advances in Physical and Inorganic Chemistry* .. .. 6th Ed. 1930
System of Bacteriology in Relation to Medicine. Vol. IX. 1931
Thomson, A., Miles, A., and Wilkie, D. P. H. *Manual of Surgery*, 3 vols. .. .. 8th Ed. (5) 1931
Tuberculosis : its Treatment and Cure with the Help of Umckaloabo (Stevens). By an English Physician .. —
Walker, J... .. .. *Organic Chemistry for Students of Medicine* 2nd Ed. 1919
Walton, A. J. .. .. *Text-book of Surgical Dyspepsias* 2nd Ed. (7) 1930
Ward, M. L. .. .. *American Text-book of Operative Dentistry* 6th Ed. (7) —
Williams, S. .. .. *Experimental History of Road Water in Wiltshire* 1731
Wilson, S. A. K. .. *Modern Problems in Neurology* .. .. (7) 1928
Winnicott, D. W. .. *Clinical Notes on Disorders of Childhood* .. 1931
Winton, F. R., and Bayliss, L. E. *Human Physiology* .. .. (8) 1930
Wood, C. A. .. .. *An Introduction to the Literature of Vertebrate Zoology* (2) 1931
Wright, S... .. .. *Applied Physiology* .. .. .. 4th Ed. 1931

TRANSACTIONS, REPORTS, JOURNALS, ETC.

Annals of the Pickett-Thomson Research Laboratory. Vol. 6. December, 1930 .. .. (7) 1930
Glasgow Royal Maternity and Women's Hospital—Medical Report for 1930 .. .. (4) 1931
International Health Year Book, 1929 .. .. (7) 1929
London School of Hygiene and Tropical Medicine—Collected Addresses and Laboratory Studies. Vol. 6 .. .. (6) 1929–30
Progressive Medicine. Vol. 3. September, 1931 1931
Royal College of Surgeons of England Calendar, 1931 .. .. (9) 1931
Saint Thomas's Hospital Reports. Vol. 53 1929
Surgical Clinics of North America, August, Vol. 2, Nos. 4 and 5. August and October, 1931 1931

Local Medical Notes

EXAMINATION RESULTS.

University of Bristol.—Students of the University have recently passed the following examinations :—

M.B., Ch.B.—*Supplementary First Examination. Pass :* J. C. D. Harris, Ursula G. Hewitt. *In Chemistry (Completing Examination) :* G. W. Vowles.

M.B., Ch.B. (*Part I.*).—*Final Examination. Pass :* S. B. Adams, A. J. Board, Violet Fry, Winifred M. Hill (with distinction in Materia Medica, Pharmacy, Pharmacology and Therapeutics), Aldyth D. Jones, Gwladys R. Llewelyn (with distinction in Pathology), H. R. Pearse (with distinction in Pathology, Forensic Medicine and Toxicology), Frances E. Powell, Mary G. Thomas.

M.B., Ch.B. (*Part II.*).—*Final Examination. Pass :* F. W. A. Fosbery, J. A. Kersley, A. J. B. Miall, S. C. Wake. *In Group I. (Completing Examination) :* O. J. P. Bollon. *In Group I. only :* A. C. Price.

L.D.S.—*Supplementary Preliminary Science Examination. Pass : In Physics and Biology only :* D. E. Royal.

L.D.S.—*Final Professional Examination. Pass :* N. Miller, H. L. Saunders.

L.D.S.—*Second Professional Examination. Pass : In Dental Mechanics and Dental Metallurgy (Completing Examination) :* I. R. R. Eskell. *In Dental Materia Medica (Completing Examination) :* R. L. Royal. *In Dental Mechanics and Dental Metallurgy only :* J. H. G. Smith.

Conjoint Board.—M.R.C.S., L.R.C.P. *Pass : In Elementary Biology :* Myrtle M. Baxter, Susie C. Sampson. *In Chemistry and Physics :* M. Thompson. *In Anatomy and Physiology :*

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